Virus Mania  Torsten Engelbrecht

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Foreword
The Content of This Book Has To Be Read, Quickly and Worldwide
The book Virus Mania by Torsten Engelbrecht and Claus Kohnlein presents a tragic message that will, hopefully, contribute to the re-insertion of ethical values in the conduct of virus research, public health policies, media communications, and activities of the pharmaceutical companies. Obviously, elementary ethical rules have been, to a very dangerous extent, neglected in many of these fields for an alarming number of years.

When American journalist Celia Farber courageously published, in Harpers Magazine (March 2006) the article Out of control-AIDS and the corruption of medical science, some readers probably attempted to reassure themselves that this corruption was an isolated case. This is very far from the truth as documented so well in this book by Engelbrecht and Kohnlein. It is only the tip of the iceberg. Corruption of research is a widespread phenomenon currently found in many major, supposedly contagious health problems, ranging from AIDS to Hepatitis C, Bovine spongiform encephalopathy (BSE or mad cow disease), SARS, Avian flu and current vaccination practices (human papillomavirus or HPV vaccination).

In research on all of these six distinct public health concerns scientific research on viruses (or prions in the case of BSE) slipped onto the wrong track following basically the same systematic pathway. This pathway always includes several key steps: inventing the risk of a disastrous epidemic, incriminating an elusive pathogen, ignoring alternative toxic causes, manipulating epidemiology with non-verifiable numbers to maximize the false perception of an imminent catastrophe, and promising salvation with vaccines. This guarantees large financial returns. But how is it possible to achieve all of this? Simply by relying on the most powerful activator of human decision making process, i.e. FEAR!

We are not witnessing viral epidemics; we are witnessing epidemics of fear. And both the media and the pharmaceutical industry carry most of the responsibility for amplifying fears, fears that happen, incidentally, to always ignite fantastically profitable business. Research hypotheses covering these areas of virus research are practically never scientifically verified with appropriate controls. Instead, they are established by consensus. This is then rapidly reshaped into a dogma, efficiently perpetuated in a quasi-religious manner by the media, including ensuring that research funding is restricted to projects supporting the dogma, excluding research into alternative hypotheses. An important tool to keep dissenting voices out of the debate is censorship at various levels ranging from the popular media to scientific publications.

We haven’t learnt well from past experiences. There are still many unanswered questions on the causes of the 1918 Spanish flu epidemic, and on the role of viruses in post-WWII polio (DDT neurotoxicity?). These modern epidemics should have opened our minds to more critical analyses. Pasteur and Koch had constructed an understanding of infection applicable to several bacterial diseases. But this was before the first viruses were actually discovered. Transposing the principles of bacterial infections to viruses was, of course, very tempting but should not have
been done without giving parallel attention to the innumerable risk factors in our toxic environment; to the toxicity of many drugs, and to some nutritional deficiencies.

Cancer research had similar problems. The hypothesis that cancer might be caused by viruses was formulated in 1903, more than one century ago. Even today it has never been convincingly demonstrated. Most of the experimental laboratory studies by virus-hunters have been based on the use of inbred mice inbred implying a totally unnatural genetic background. Were these mice appropriate models for the study of human cancer? (We are far from being inbred!) True, these mice made possible the isolation and purification of RNA tumor viruses, later renamed retroviruses and well characterized by electron microscopy. But are these viral particles simply associated with the murine tumors, or are they truly the culprit of malignant transformation? Are these particles real exogenous infective particles or endogenous defective viruses hidden in our chromosomes? The question is still debatable. What is certain is that viral particles similar to those readily recognized in cancerous and leukemic mice have never been seen nor isolated in human cancers.

However, by the time this became clear, in the late 1960s; viral oncology had achieved a dogmatic, quasi-religious status. If viral particles cannot be seen by electron microscopy in human cancers, the problem was with electron microscopy, not with the dogma of viral oncology! This was the time molecular biology was taking a totally dominant posture in viral research. Molecular markers for retroviruses were therefore invented (reverse transcriptase for example) and substituted most conveniently for the absent viral particles, hopefully salvaging the central dogma of viral oncology. This permitted the viral hypothesis to survive for another ten years, until the late 1970s, with the help of increasingly generous support from funding agencies and from pharmaceutical companies. However by 1980 the failure of this line of research was becoming embarrassingly evident and the closing of some viral oncology laboratories would have been inevitable, except that.

Except what? Virus cancer research would have crashed to a halt except that, in 1981, five cases of severe immune deficiencies were described by a Los Angeles physician, all among homosexual men who were also all sniffing amyl nitrite, were all abusing other drugs, abusing antibiotics, and probably suffering from malnutrition and STDs (sexually transmitted diseases). It would have been logical to hypothesize that these severe cases of immune deficiency had multiple toxic origins. This would have amounted to incrimination of these patients life-style. Unfortunately, such discrimination was, politically, totally unacceptable.

Therefore, another hypothesis had to be found-these patients were suffering from a contagious disease caused by a new retrovirus! Scientific data in support of this hypothesis was and, amazingly enough, still is totally missing. That did not matter, and instantaneous and passionate interest of cancer virus researchers and institutions erupted immediately. This was salvation for the viral laboratories where AIDS now became, almost overnight, the main focus of research. It generated huge financial support from Big Pharma, more budgets for the CDC and NIH, and nobody had to worry about the life style of the patients who became at once the innocent victims of this horrible virus, soon labeled as HIV.

Twenty-five years later, the HIV 1 AIDS hypothesis has totally failed to achieve three major
goals in spite of the huge research funding exclusively directed to projects based on it. No AIDS cure has ever been found; no verifiable epidemiological predictions have ever been made; and no HIV vaccine has ever been successfully prepared. Instead, highly toxic (but not curative) drugs have been most irresponsibly used, with frequent, lethal side effects. Yet not a single HIV particle has ever been observed by electron microscopy in the blood of patients supposedly having a high viral load! So what? All the most important newspapers and magazine have displayed attractive computerized, colorful images of HIV that all originate from laboratory cell cultures, but never from even a single AIDS patient. Despite this stunning omission the HIV I AIDS dogma is still solidly entrenched. Tens of thousands of researchers, and hundreds of major pharmaceutical companies continue to make huge profits based on the HIV hypothesis. And not one single AIDS patient has ever been cured.

Yes, HIV I AIDS is emblematic of the corruption of virus research that is remarkably and tragically documented in this book. Research programs on Hepatitis C, BSE, SARS, Avian flu and current vaccination policies all developed along the same logic, that of maximizing financial profits. Whenever we try to understand how some highly questionable therapeutic policies have been recommended at the highest levels of public health authorities (WHO, CDC, RKI etc.), we frequently discover either embarrassing conflicts of interests, or the lack of essential control experiments, and always the strict rejection of any open debate with authoritative scientists presenting dissident views of the pathological processes. Manipulations of statistics, falsifications of clinical trials, dodging of drug toxicity tests have all been repeatedly documented. All have been swiftly covered up, and none have been able to, so far, disturb the cynical logic of today’s virus research business. The cover-up of the neurotoxicity of the mercury containing preservative thimerosal as a highly probable cause of autism among vaccinated children apparently reached the highest levels of the US government.

Virus Mania is a social disease of our highly developed society. To cure it will require conquering fear, fear being the most deadly contagious virus, most efficiently transmitted by the media.

Introduction
The founding of The Royal Society in 1660 caused a tectonic shift in Western medicine. A group of British scientists decided that what counts is the experimental proof not speculative fantasy, superstition and blind faith. The Royal Society called this basic research principle nullius in verba, which essentially means Don’t just trust what someone says. In that era, it was still common to accuse women of witchcraft in the name of God and turn them at the stake, or to subjugate entire peoples such as the Aztecs or Mayans to Western ideologies. Setting a standard of scientific proof marked the end of the dark ages and had enormous long-term consequences.

Today, considering ourselves enlightened and in the safe hands of our high-tech scientific culture, we look back with misgivings and great discomfort at the abuses of power that occurred in such draconian times. Indeed, the dream that science promises with its principle of proof—namely to free people from ignorance, superstition, tyranny, and not least from physical and psychological suffering—has, in many cases, particularly in wealthy countries, become a reality. Airplanes, tractors, computers, bionic limbs—all these achievements are the product of scientific
research. Like our modern legal system, bound by the principle of evidence, science recognizes only one guiding principle: provable fact.

Our enthusiasm for scientific achievements has risen immeasurably. We have granted a godlike status to researchers and doctors, who still had the status of slaves in ancient Rome and even until the early 20th century were mostly poor and powerless. Because of this status, we continue to perceive them as selfless truth seekers.

The English biologist Thomas Huxley, a powerful supporter of Charles Darwin and grandfather of the author Aldous Huxley (Brave New World, 1932), described this phenomenon as early as the late 19th century, when he compared sciences growing authority to the Church’s position of power. For this, he coined the term Church Scientific.

Today’s enlightened civilized individual believes so firmly in the omnipotence of scientists that they no longer question the evidence for certain hypotheses or even whether they make sense. Instead, citizens rely on the latest sensationalized media coverage churned out in daily newspapers and TV newscasts about world-threatening viral epidemics (Avian Flu, SARS, AIDS, etc.). For many decades, the media (and scientific reporters above all) have intently cultivated friendly relationships with researchers in the drive to scoop their competitors for provocative headlines. We scientific reporters all too often serve as living applause for our subject, New York Times reporter Natalie Angier says critically about her profession. Sometimes we write manuscripts that sound like unedited press releases.

Journalists usually assume that scientists engage in rigorous studies and disseminate only provable facts—and that rare instances of fraud will quickly be driven out of the hallowed halls of research. It’s an ideal picture, but one that has nothing to do with reality. Uncountable billions of dollars are transformed into scientific hypotheses, which are ultimately packaged and hawked by pharmaceutical companies, researchers, health advocates and journalists alike as the ultimate conclusions of truth. In actuality, these theories are often mere speculation, proven false and years later, finally discarded.

The more willing the people are, the more promises must be made, warned Erwin Chargaff as early as 1978. A quick route to long life, freedom from all diseases, a cure for cancer—soon, perhaps the elimination of death—and what then asked the co-founder of biochemical research and gene-technology, and a repeatedly decorated professor at Columbia University’s Biochemical Institute in New York. But no singer would ever have to promise to make me a better person if I would just listen to her trills.

Since the end of the 1970s, this situation has dramatically worsened. Just as in politics and economics, we in research are also bombarded, saturated, harried by fraud, writes renowned science historian Horace Judson, whose analyses are corroborated by a number of relevant studies. From a global viewpoint, there is corruption at all levels of the public health service, from health ministry’s to patients—and there are almost no limits to criminal imagination, maintains Transparency International, an institution for protection against corruption, in its annual Global Corruption Report 2006 (focus on health services).
Use multiple endpoints (survival time, reduction of blood pressure, etc.) in the trial and select for publication those that give favorable results. Conduct trials that are too small to show differences from competitor drugs. Do multicenter trials and select for publication results from centers that are favorable. Source: Smith, Richard, Medical Journals Are an Extension of the Marketing Arm of Pharmaceutical Companies Medicine, May 2005.

A close look at this data reveals that our scientific culture is ruled by secretiveness, privilege-granting, lack of accountability, and suffers from a blatant lack of monitoring, as well as from the prospects that these companies and researchers will make exorbitant profits. All of these questionable factors contribute to the potential for researcher bias and fraud, jeopardizing the scientific proof principle introduced in the 17th century. Judson paints a dark picture of [biomedical] science today, but we may see far darker days ahead as proof and profit become inextricably mixed, warns the medical publication Lancet.

Even when one theoretically assumes ideal researchers and ideal studies, it must be emphasized that medicine remains (is still) a science of uncertainties, expressed William Osler (1849 - 1919), regarded as the father of modern medicine. Nothing has changed. Donald Miller, Professor of Surgery at the University of Washington, warns that with today’s medical research, scientific standards of proof are not uniform and well defined, in contrast to legal standards. Standards of measurement, ways of reporting and evaluating results, and particular types of experimental practices vary. Science prizes objective certainty. But science does not uniformly adhere to this standard. Subjective opinions and consensus among scientists often supersede the stricture of irrefutability.

To effectively combat this systemic problem, much would be gained if it were compulsory to have certain studies replicated, thus reviewing them for their soundness. But, according to Judson, replication, once an important element in science, is no longer an effective deterrent to fraud because the modern biomedical research system is structured to prevent replication—not to ensure it. Such verification is unattractive, because it doesn’t promise gigantic profits, but might only produce similar results to the original research, which is unlikely to be published by a medical journal. From time to time, these reviews are carried out, with stunning results.

At the beginning of 2005, an investigation disclosed a severely flawed study leading to the approval of Viramune, a globally-touted AIDS medicine ranked among the top sellers of pharmaceutical giant Boehringer Ingelheim (the drug Viramune brings in approximately $300 million annually). The follow-up investigation found that records of severe side effects including deaths were simply swept under the carpet.

At the same time, chief investigator Jonathan Fishbein was greatly hindered, from the highest levels of the National Institutes of Health, in his bid for clarification. The medical system, according to Fishbein, is shaped more by politics of interest, partisanship and intrigue than by sound science. Fishbein called the government’s AIDS research agency a troubled organization, referring to an internal review that found its managers have engaged in unnecessary feuding, sexually explicit language and other inappropriate conduct.
How far this can go becomes apparent when research produced by individual scientists is placed under the microscope. The South Korean veterinarian Hwang Woo Suk, for example, published a paper in Science in May 2005 in which he described how he had extracted human stem cells from cloned embryos for the first time. The work was celebrated as a global sensation and Hwang as a cloning pioneer. But at the end of 2005, it was discovered that Hwang had completely forged his experiments.

The medical field is ultimately about illness, dying and death: Naturally, these experiences involve a complex and nuanced range of emotions for individuals, their loved ones and doctors. The process makes us extremely receptive to a belief in salvation through miracle treatments. In this, researchers and physicians take over the roles of priests; the white smock has merely replaced the black robes and black wigs physicians used to wear. These white knights proclaim their healing messages, and of course require victims to carry out their research with billions of dollars of government and taxpayer funded dollars. Indeed, so profound is our belief in the cures of science that it has become the new secular theology of the 20th century, according to American media scientist Michael Tracey. This belief is so inherent within us that we construct any problem, grievance, pain, or fear in conceptual terms that not only allow us to seek the cure, but demand that we do so.

At the heart of this web of feelings and wishes are the fantasies of almightiness that further prop up the medical-industrial complex, that ever more powerful part of the global economy consisting of pharmaceutical companies worth billions, their lobbyists and spin doctors, and an immense army of highly-paid researchers and doctors. In the process, we’ve turned our bodies into vehicles of consumerism, internalizing a highly-questionable promise inherent to this industry: Science can conquer terrible and puzzling diseases-just like we conquered the moon-if it is just given enough money.

To avoid any misunderstandings: medicine has made tremendous achievements. This applies first and foremost to reparative medicine such as accident surgery, organ transplants or laser eye surgery. But, the various perils of modern medicine are all-too evident in the ever-expanding field of so-called preventive and curative treatments, particularly the growing arsenal of pharmaceutical drugs-in other words, medicine that purports to be able to heal.

Take cancer, for example. In 1971, US President Richard Nixon at the behest of public health officials (and above all, virologists), declared a War on Cancer. The medical establishment vowed there would be a cure at hand by 1975. But we are still waiting. And there is no evidence of the way cancer comes into being, according to German Cancer Research Center.

Mainstream cancer theories also show blatant contradictions. Despite this, hundreds of billions of dollars have already flowed into a completely one-sided cancer research focused on wonder-drug production. Above all, this set-up grants pharmaceutical companies, researchers and doctor’s gigantic profits. In contrast, even plausible alternative theories (which may be less profitable, because they focus on lifestyle and environmental factors and not only on fatefully appearing genes and viruses as causes) remain almost completely disregarded.
For instance, although official cancer theories assume that a third of cancer cases could be prevented through a change of diet (above all more fruit and vegetables and less meat), cancer expert Samuel Epstein points out that the American National Cancer Institute spent just $1 million—that is 0.02 percent of its $4.7 billion budget in 2005-on education, press work and public relations to encourage eating fruit and vegetables to prevent cancer.

At the same time, the number of people who die from non-smoking cancers has noticeably increased since Nixon’s call to battle (even, it is worth noting, when one takes into consideration that people on average have become older). Today in Germany alone, 220,000 people die from this terrible disease annually; in the USA there are almost 600,000 cancer deaths each year. The situation doesn’t look any better for other widespread illnesses such as diabetes, heart disease, high blood pressure, or rheumatism. In spite of exorbitant research budgets, the development of a cure is unforeseeable. Cortisone, for instance, does help to alleviate acute rheumatic or allergic discomfort—but only during cortisone therapy. If treatment is discontinued, suffering returns. At the same time, cortisone, which also finds plenty of use in the treatment of viruses, is, like most reputed miracle cures (magic bullets), connected with severe side effects. Vera Sharav of the New York City-based Alliance for Human Research Protection (AHRP), an organization that fights for independent and ethically responsible medical science, warns that often enough, the medications are so toxic that they produce precisely the diseases against which, as the pharmaceutical manufacturers advertising messages aim to convince us, they are supposed to be so active. And then, new preparation after new preparation is given.

As relevant studies reveal, drug toxicities are so severe that the American health industry’s pill craze is responsible for about 800,000 deaths each year, more than any illness (including cancer and heart attack). And in Germany, tens of thousands of people are estimated to die each year due to improper treatment and prescription of incorrect medications (there are no exact figures because certain interest groups have successfully resisted the collection of the relevant information).

The fact that a society calling itself enlightened is nevertheless dominated by the belief that there is a healing pill for every little ache and pain or serious complaint is substantially due to the persuasive craftiness of Big Pharma. Pharmaceutical companies operating in the US spend approximately a third of their expenses on marketing, which means that $50 billion per year is merely invested in advertising their preparations as miracle cures to doctors, journalists, consumers and politicians.

With this, they have extended their sphere of influence in a most alarming way to include institutions like the World Health Organization (WHO), the Food and Drug Administration (FDA), as well as the US National Institutes of Health (NIH), the independence and integrity of which is particularly important.

A study published in the Journal of the American Medical Association (JAMA) in April 2006, showed that conflicts of interest at the FDA are widespread. It was shown that in 73% of meetings, at least one member of the consulting team in question yielded conflicts of interest: being remunerated by Big Pharma, for instance, through consultation fees, research contracts or
grants, or stock ownership or options. In nearly a quarter of contracts and grants, for example, sums of more than $100,000 changed hands. The study found that these conflicts of interest influenced voting behavior. When panel members with conflicts of interest were excluded from voting, the judgment of the product in question was much less favorable. And even though these conflicts of interest were so extensive, panel members with relevant conflicts of interest were disqualified in only 1% of cases.

Big Pharma money and advertising not only influence the perception of illness, the demand for drugs, and the practice of medicine, but government budgets, including health service and oversight agencies have become dependent on Big Pharma money, says Vera Sharav of the AHRP. An out of the box analysis opened our eyes to a fundamental conflict of interest that has never been discussed. Public health policies are not merely influenced by Big Pharma; they are formulated so as to increase industry’s profits because government budgets are tied to this industry’s profits. In this context, a decisive event occurred in 1992 when the US Congress waved through the Prescription Users Fees Act (PDUFA), which established the fast track drug approval service. According to Sharav, the FDA has received $825 million in industry user fees, and other government agencies have similarly become financially dependent on Big Pharma. The issue stirred up so much controversy that the British Parliament also opened an extensive investigation. Their conclusions: The pharmaceutical industry’s corrupt practices and its massive influence upon parliaments, authorities, universities, health professionals and the media were sharply criticized.

In fact, if prescription medicines are so good, why do they need to be pushed so hard? asks Marcia Angell, former Editor in Chief of the well-known New England Journal of Medicine (NEJM). Good drugs don’t have to be promoted. Her opinions are as simple as they are revealing, but unfortunately they don’t register in the consciousness of the modern believer in science. Our society that considers itself particularly enlightened has become senselessly overmedicated.

This pill-mania exists because we have a distorted comprehension of what causes diseases—a comprehension that has been able to lodge itself firmly in our thought processes over a period of more than 100 years. To understand this, one must look back to the middle of the 19th century, when a true paradigm shift in the way we see disease occurred. There was an about-turn, away from a complex, holistic view concerning how diseases originate, to a monocausal and one-dimensional mindset, to use a term from philosopher Herbert Marcuse. Through this, a false awareness arose which is immune to its falseness because the dimensions of self-criticism and the ability to look in various alternative directions is missing.

This paradigm shift is largely due to the fact that from approximately the 16 century, in the course of the Enlightenment, the natural sciences began to develop rapidly, and put the population under their spell with descriptions of very specific phenomena. One need only remember the tremendous achievements of the English physicist Isaac Newton, who described gravitation; or the invention of the steam locomotive or even the printing press. But in the euphoric exuberance of progress, particularly from the middle of the 19 century, this thought pattern of specificity that very particular chemical or physical phenomena have very specific
causes was simply transferred to the medical sciences. Many researchers and interest groups didn’t even consider if this actually made sense.

The dogma of a single cause for diseases was decisively shaped by microbiology, which became predominant at the end of the 19th century, declaring specific microorganisms (viruses, bacteria, fungi) to be the causes of very definite diseases; including mass epidemics such as cholera and tuberculosis. The founders of microbe theory, researchers Louis Pasteur and Robert Koch, ascended in their lifetimes to the heights of medicine's Mount Olympus.

And so with the microbe theory, the cornerstone was laid for modern biomedicines basic formula with its monocausal-microbial starting-point and its search for magic bullets: one disease, one cause, one cure, writes American sociology professor, Steven Epstein. From the end of the 19 century, the hunt for microbes increasingly provided the thrill, and the same admiration that physicists and chemists had earlier garnered (as in Paris in 1783, when the brothers Montgolfier performed the miracle of launching a hot air balloon into the sky).

But as fascinating as this conception of a single cause is, it has very little to do with the complex workings of the human body. A significant majority of diseases have far more than just one cause, so the search for the single cause of disease, and by extension for the one miracle-pill, will remain for them a hopeless undertaking. This is particularly true in microbiology, a scientific No Man’s Land, as the American magazine The New Yorker fittingly described it. The field is becoming ever more complex and incomprehensible, as further research penetrates the seemingly infinite microcosmic mini-worlds of cellular components, molecules and microbes.

Bacteria, fungi and viruses are omnipresent-in the air, in our food, in our mucous membranes-but we aren’t permanently sick. When a disease generally held to be contagious breaks out, only some individuals become sick. This is clear evidence that microbes, whatever potential they may have to make you sick, cannot be the lone cause of disease.

Pasteur himself admitted on his deathbed: The microbe is nothing, the terrain is everything. And indeed, even for mainstream medicine, it is becoming increasingly clear that the biological terrain of our intestines-the intestinal flora, teeming with bacteria-is accorded a decisive role, because it is by far the body’s biggest and most important immune system. A whole range of factors (in particular nutrition, stress, lack of activity, drug use, etc.) influence intestinal flora, so it has a decisive influence on all sorts of severe or less serious illnesses.

But it is not just this large oversimplification that calls for opposition to the microbe theory. Under closer examination, fundamental assumptions of microbe theory also emerge as pure myth. Edward Kass, professor of medicine at Harvard University, made this the subject of his opening address at a conference of the American Society for Infectious Diseases in 1970. US citizens were becoming increasingly critical of the Vietnam War and many people in the USA began to rebel against the establishment. Maybe this zeitgeist spurred Kass to address these issues openly, although they may have stood in glaring opposition to the views of most of his listeners.

Kass argued that medical scientists and microbe hunters were not the ones to be praised for
stemming the flow of mass diseases like tuberculosis, diphtheria, measles, whooping cough or pulmonary infections. The data unquestionably shows that death rates for these so-called infectious diseases had noticeably decreased from the middle of the 19th century; long before microbe hunters and the medical establishment became active. The monumental accomplishment of pushing back diseases and raising life expectancy is primarily due to an improvement in general standards of living (improved nutrition, construction of water purification plants, etc.), which gained momentum in industrialized countries precisely in the mid-19th century.

This also explains why deaths from so-called infectious diseases have become a rarity in affluent societies (in wealthy countries, they make up less than 1% of all mortalities). Yet, in poor third-world regions like Africa, where every third person is malnourished, these same diseases (tuberculosis; leprosy, etc.) that wealthy countries fought during times of recession run rampant. The excessive panic-like fear, which so easily consumes members of affluent societies when the media stokes the flames of the viral-epidemic panic, can in this context, only be described as irrational.

Recently, headlines on avian flu and the SARS virus have dominated global reports, but the world is also exposed to horror scenarios about hepatitis C, AIDS, Ebola and BSE. These shocking media reports totally overlook the fact that the existence and pathogenic effects of all these allegedly contagious and even fatal viruses-avian flu, HSN1, HN etc.-have never been proven. A glaring paradox is that very few people actually die from these purported large new epidemics. Strictly speaking, these epidemics are not epidemics whatsoever.

No scientists have even seen the avian flu virus HSN1 in full (with its complete genetic material and virus shell); we don’t even know if it could be dangerous to humans, or if it could trigger the already widely reported global pandemic; something that mainstream researchers also admit. And despite this Jack of proof, Reinhard Kurth, director of Germany’s Robert Koch Institute, which is responsible for microbe epidemics, does not shy from warning that HSN1 potentially threatens all of humanity. There is also discrepancy between speculation and existing facts in the BSE epidemic, which has yet to present us in Germany with a single clinical case of the disease, only animals that have tested positive for the virus.

With regard to hepatitis C, we are still waiting for the predicted epidemic of liver cirrhosis (serious liver damage). Since the 1980s, no more than a few hundred people die in Germany each year from so-called AIDS, according to official statistics. And what about the horrifying figures of x-million infected with HIV in Africa and other developing countries? This is primarily due to the redefinition of patients who suffer from conventional diseases like tuberculosis or leprosy as AIDS patients. The threat of SARS is similarly over hyped: In the first nine months (November 2002 - July 2003) after the alleged discovery of the SARS virus at the end of 2002, the World Health Organization found only 800 probable SARS deaths.

Years from now, people looking back at us will find our acceptance of the HIV theory of AIDS as silly as we find the leaders who excommunicated Galileo, just because he insisted that the earth was not the center of the universe, predicts Kary Mullis, one of the most significant Nobel laureates of the 20th century. It has been disappointing that so many scientists have absolutely refused to examine the available evidence in a neutral, dispassionate way, regarding whether
HIV causes AIDS. This breaking of the fundamental principles in scientific research also applies to other new alleged epidemics like hepatitis C, SARS, avian flu, cervical cancer, Ebola, and BSE.

Mullis words come from his article titled, The Medical Establishment vs. the Truth. In it, he discusses how the entire virus-busting industry plies its dogmas, declaring them to be eternal truths, without the support of factual evidence. Of course, this helps to secure the gigantic research budgets and profits of pharmaceutical groups and top scientists.

Between 1981 and 2006, US taxpayers alone shelled out $190 billion for AIDS research focused almost exclusively on the deadly virus hypothesis and the development of treatment drugs. Yet the growing list of medications haven’t demonstrably extended the life of a single patient, and a cure is nowhere in sight.

The same strategy has been employed with Tamiflu flu medication, which has serious side effects, yet, thanks to skillful public relations work, support of the WHO and the medias avian flu fear mongering, this drug mutated in a short time from shelf warmer to cash cow.

While pharmaceutical groups and top researchers cash in and the media drive their circulation ratings sky high with sensationalized headlines, citizens must foot a gigantic bill without getting what is necessary: enlightenment over the true causes and true solutions. So what are dedicated clinicians to do? asks John Abramson of Harvard Medical School. The first step is to give up the illusion that the primary purpose of modern medical research is to improve Americans health most effectively and efficiently. In our opinion, the primary purpose of commercially-funded clinical research is to maximize financial return on investment, not health.

This books central focus is to steer this discussion back to where, as a scientific debate, it belongs: on the path to prejudice-free analysis of facts. To clarify one more time, the point is not to show that diseases like cervical cancer, SARS, AIDS or hepatitis C do not exist. No serious critic of reigning virus theories has any doubt that people or animals (as with avian flu) are or could become sick (although many are not really sick at all, but are only defined as sick, and then are made sick or killed). Instead, the central question is: What really causes these diseases known as cervical cancer, avian flu, SARS, AIDS and hepatitis C? Is it a virus? Is it a virus in combination with other causes? Or is it not a virus at all, but rather something very different?

We will embark on a detailed examination of the hypotheses of science, politics and the media elite, looking at all of the available evidence. At the same time, alternative explanations or causes will be described: substances like drugs, medicines, pesticides, heavy metals or insufficient nutrition. All these factors can severely damage or even completely destroy the immune system— and their devastating effects can be encountered in the victims hastily branded with a diagnosis of cervical cancer, avian flu, SARS, AIDS or hepatitis C. Ultimately they are victims of complex, broad socio-economic and political forces and further marginalized and degraded by a profession that pledges to do no harm.

Chapter 1 explains what microbes (bacteria, fungi, viruses) actually are, and what role they play in the complete cycle of life and the ways in which the medical establishment and the media
have turned these microbes into our worst enemies. In Chapter 2, well travel from the middle of the 19th century until modern times, in order to separate myth from reality in microbe theory. Louis Pasteur and Robert Koch rose to become medicines shining lights, but we cannot leave them out of this analysis since they were certainly not immune from lying and deception. Nor will we shy away from the question of whether polio is a viral disease or if poisons like pesticides have not made at least their contribution to the destruction of the spinal nerves that is so typical of this disease.

With this background knowledge, we dive into the past three decades: into the time of modern virus research. Chapter 3 thus begins with the history of HIV I AIDS, which arrived in the early 1980s, triggering an almost unprecedented mass panic that continues to this day. And now the whole world also seems to accept that Hepatitis C, BSE, SARS, avian flu and cervical cancer are also triggered by a causative agent (pathogen). In Chapters 4 through 8 we will see that these statements do not hold up and those other explanations make more sense?

Chapter 1
People are very susceptible to the idea that certain microbes act like predators, stalking our communities for victims and causing the most serious illnesses like SARS (pulmonary infection) or hepatitis C (liver damage). Such an idea is thoroughly simple, perhaps too simple. As psychology and social science have discovered, humans have a propensity for simplistic solutions, particularly in a world that seems to be growing increasingly complicated. It also allows for a concept of the enemy at the gates allowing individuals to shift responsibility for their illnesses to a fungus, a bacteria or a virus. Man prefers to perish rather than change his habits! the author Leo Tolstoy once said.

But this scapegoat thinking has often led humanity astray, be it in personal life, in science or in politics. Fishermen and politicians both earnestly assert that seals and dolphins contribute to the depletion of ocean fish stocks. So, each year in Canada, one hundred thousand seals-often just a few days old-are battered to death, while every autumn in Japan, thousands of dolphins are hacked apart while still alive.

But in their blind hate for the animals, the slaughterers completely overlook the fact that it is their own species-Homo sapiens-is responsible for the state of our oceans and that through massive overexploitation and high-technology catch methods, we have plundered the worlds fish stocks. A German-Canadian study that appeared in Nature in 2003, found that industrialized fishing has dramatically reduced the stocks of predators like tuna and swordfishes, marlins, cod, halibut, ray and flounder in the world’s oceans since the beginning of commercial fishing in the 1950s-by no less than 90%.

Our modern concept of the lethal microbes similarly avoids the big picture issues. Some can be harmful; nevertheless, it is negligent to ignore the role individual behaviors (nutrition, drug consumption, etc.) play instead of simply pointing a finger at these microorganisms. Whether the method of treatment affects the animal predators in the wilderness or the bacteria in the gut, it is always risky to tamper with the natural balance of forces in nature, writes microbiologist and Pulitzer Prize winner Rene Dubos.
Medical and biological realities, like social ones, are just not that simple. Renowned immunology and biology professor Edward Golub's rule of thumb is that, if you can fit the solution to a complex problem on a bumper sticker, it is wrong! I tried to condense my book The Limits of Medicine: How Science Shapes Our Hope for the Cure to fit onto a bumper sticker and couldn’t.

The complexities of the world—and above all, the living world—might seem too difficult for any one individual to grasp with even approximate comprehension. Informing ourselves on economics, culture, politics and medical science seems incredibly daunting. Man is not an Aristotelian god that encompasses all existence; he is a creature with a development who can only comprehend a fraction of reality, writes social psychologist Elisabeth Noelle-Neumann. Supposed experts are no exception. Most doctors themselves, for instance, have hardly more than a lay understanding of the concepts that loom on the horizons of molecular biology, including research into microbes and their role in the onset of diseases.

Correspondingly, if you asked most doctors to define the unmistakable characteristics of retroviruses (HIV, for example, is claimed to be one), they most likely shrug their shoulders or throw out a bewildering cryptic response. Another challenge for many doctors would be a description of how the polymerase chain reaction (PCR) functions, even though it developed into a key technology in molecular biology in the 1990s, and is brought up again and again in connection with the alleged discovery of the so-called avian flu virus HSN1 (on PCR, see chapter 3, about the miracle weapons of the epidemic inventors).

Medicine Presents a Distorted Picture of Microbes
Ignorance and the desire for oversimplification are root problems in medical science. As early as 1916, the philosopher Ludwig Wittgenstein remarked in his diary: Humanity has always searched for a science in which simplex sigillum verist, essentially meaning, simplicity is a mark of truth. And microbe theory fits exactly into this scheme: one disease, one agent as cause—and ultimately, one miracle pill or vaccine as a solution.

But this oversimplification belies the goings-on in the invisible micro-world of cells and molecules. The living world—on both a small and large scale—is just much more complicated than medical science and the media lets on. For this reason, as biochemist Erwin Chargaff points out, The attempt to find symmetry and simplicity in the worlds Jiving tissue has often led to false conclusions. There are even a few people who believe that what is now called molecular biology encompasses all life sciences. But that is not the case, except on a superficial level: everything we can see in our world is somehow made up of molecules. But is that all? Can we describe music by saying that all instruments are made of wood, brass, and so on, and that because of that they produce their sounds?

Biology—the science of life—isn’t even capable of defining its own object of research: life. We do not have a scientific definition of life, as Erwin Chargaff states. And indeed, the most precise tests are carried out on dead cells and tissues. This phenomenon is particularly virulent in bacterial and viral research (and in the whole pharmaceutical development of medicines altogether) where laboratory experiments on tissue samples which are tormented with a variety
of often highly reactive chemicals allow few conclusions about reality. And yet, conclusions are constantly drawn and then passed straight on to the production of medications and vaccines. It’s ultimately impossible to find out exactly everything that microbes get up to on a cellular and molecular level in living people or animals. To do this, you would have to chase every single microbe around with mini-cameras. And even if it were possible, you’d merely have little pieces of a puzzle, not an intricate blueprint of the body in its entirety. By focusing on microbes and accusing them of being the primary and lone triggers of disease, we overlook how various factors are linked together, causing illness, such as environmental toxins, the side effects of medications, psychological issues like depression and anxiety and poor nutrition.

If over a longer period of time, for instance, you eat far too little fresh fruits and vegetables, and instead consume far too much fast food, sweets, coffee, soft drinks, or alcohol (and along with them, all sorts of toxins such as pesticides or preservatives), and maybe smoke a lot or even take drugs like cocaine or heroin, your health will eventually be ruined. Drug-addicted and malnourished junkies aren’t the only members of society who make this point clear to us. It was also tangibly presented in the 2004 film Super Size Me, in which American Morgan Spurlock—the film’s director and guinea pig rolled into one—consumed only fast food from McDonald’s for 30 days. The result: Spurlock gained 12 kg, his liver fat values were equivalent to those of an alcoholic, his cholesterol increased, he became depressed, suffered from severe headaches and erectile dysfunction.

Despite its drastic effects, people still become addicted to this protein and fat containing and simultaneously nutrient-deficient foodstuff. Certainly that has something to do with the fact that fast-food corporations with an annual advertising budget of over $1.4 billion, purposefully and successfully target the smallest consumers (while the US government provides an advertising budget of merely $2 million for their campaign Fruit and Vegetables—five times a day). As laboratory studies on rats and mice show, the contents of hamburgers and French fries can cause reactions in the body that are similar to that of heroin addiction, which has been proven to have a destructive effect upon the immune system. Significant components in the onset of addiction, according to researchers, are processed ingredients. A diet containing salt, sugar and fats caused the animals to become addicted to these foodstuffs, says Ann Kelley, a neurologist at the Wisconsin Medical School who observed alterations in brain chemistry in long-term test series that were similar to long-term use of morphine or heroin.

Sugar is in a position to be a gateway to other drugs, legal or illegal, according to Thomas Kroiss, president of the Austrian Society for holistic medicine. Sugar robs vitamins from the body, which influences mood as well. Although it is popular in Western cultures it doesn’t exist at all in nature, and causes an imbalance when regularly consumed.

This prompted the journal New Scientist to write that fast foods, like cigarettes, should carry a health advisory warning. But instead of providing more information and carrying out more research (not least into the influence of animal proteins on health not just those found in burgers) on the many dangers of fast foods, McDonalds continues luring children with Happy Meals and even promotes the brand by sponsoring large sporting events.

One such event was the Football World Cup 2006 in Germany, which was supposed to be all
about sport-and by extension health. To push its image as a promoter of health, the fast food giant has founded a children’s aid program, McDonalds Kinderhilfe—for sick children who, according to the fast food giant, need one thing above all: love and security. Super-celebrities such as athletes Michael Ballack, Henry Maske, Miroslav Klose and Katarina Witt, as well as supermodel Heidi Klum and the world-famous vocal trio Destiny’s Child functioned as brand-pushers. Corporate groups also receive political support. In late 2005, the EU commission announced that they wanted to loosen TV advertising regulations, making even more and more specifically targeted advertising possible, such as direct product placement during programs. If these measures had been carried out, European cultures would undoubtedly have found themselves closer to US standards—and the consumer would be even more heavily bombarded with advertising messages from the food, pharmaceutical and other multi-national industries. Such partisan politics certainly have nothing to do with targeted health precautions, although that kind of public service is so urgently needed.

Preventive health care is generally neglected by the very government-sponsored groups charged with protecting the health of citizens. A good and symbolically appropriate example of this is that these bloated bureaucracies pay little attention to intestinal function and health. Even organizations like the generally esteemed Stiftung Warentest, a German consumer protection organization still earnestly holds to the message that poor nutrition or a lifestyle that leads to constipation generally has nothing to do with intestinal bacteria; Candida fungi, for instance, can be found in every healthy intestine. And in general, shifts in the composition of the intestines microbes are merely symptoms [that is, consequences] of infections, inflammations or antibiotic treatments, but not their causes. Under normal patterns of life, the intestinal flora regulates itself on its own as soon as the cause of the disturbance has been eliminated, the researchers say. Stiftung Warentest cannot, however, furnish concrete studies that prove this.

And there is also no reason to assume that their statements are well founded. Beyond the allegedly sole causes (infections, inflammations) of a shift in the intestinal flora, of course there are many factors to consider. A large proportion of the population suffers from intestinal problems like constipation or abnormally high Candida fungus, so, it’s absurd to assume that toxins and antibiotics should pass by the intestinal flora’s composition without leaving a trace. We don’t even know precisely what a normal intestinal flora is. We’ve yet to become acquainted with all the microbes in the intestinal ecosystem, and it has also been observed that different people have very different intestinal flora. How, then, could we possibly know what normal intestinal flora looks like? Or how it constantly regulates itself toward a normal level? The individual microbe composition might be very stable, as studies suggest, but stable but doesn’t automatically mean normal or even healthy.

It is certain that artificial sugar, for example, constitutes a terrain for the wrong fungi and bacteria, says physician Thomas Kroiss. Additionally, studies document that a diet with little to no fresh (raw) food is unsuitable for maintaining a properly functioning intestinal flora. Individual behavior (nutrition, activity, stress, etc.) also influences intestinal flora, and can also lead to pathogenic Candida fungi.

In this context, it would also be interesting to discover what kind of effect an overly acidic diet has on the intestinal flora and on the health of an individual. After all, studies on animals in
factory farms show that the acids ingested with food, which are said to speed up growth in pigs or poultry, affect intestinal flora negatively.

But, how does it affect the human body?
The human body is like a forest with a buffer system of lungs, kidneys and sweat glands, by way of which superfluous acids can be released. The German Nutrition Society claims that an excessively basic diet brings no provable advantages to your health. Too much acid in the body is nothing to fear in a healthy individual, since buffer systems keep the acid-base level in blood and tissue constant. Still, the DGE cannot deliver any evidence for its claim, and it is difficult to imagine that a normal diet, that only consists of acid generating foods like meat, fish, eggs, cheese, bread, butter, refined sugar and pills and few to no base-producing foods like fruit and vegetables can leave no trace in the body.

Even if the buffer systems in a so-called healthy person (whatever that means keeps the acid-base level in the blood constant, it cannot be ruled out that tissue may be stressed or even damaged. Many experts, such as the American nutritionist Gary Tunksy are of the opinion that the fight for health is decided by the pH values. It is worth noting that cancer tissue, for instance, is extremely acidic, and it would be easy to investigate how various basic or acidic diets affect the course of the cancer-but unfortunately this doesn’t happen. The influence that nutrition has on the skeletal system, on the other hand, has been well investigated; even osteoporosis tablet manufacturers expressly indicate that one should try to avoid phosphate and foods containing oxalic acids, in other words [calcium robbers like] meat, sausages, soft drinks, cocoa or chocolate.

The intestinal flora is among the numerous factors that could take part in the onset and triggering of an illness, states Wolfgang Kruis, intestinal expert and professor of medicine in Cologne. And his colleague, researcher Francisco Guamer, adds that the intestinal flora is very significant to an individual’s health, something that has been well documented. Among other things, it is essential in providing nutrients for the development of epithelial cells. And if the intestine is disturbed, this can affect the absorption and processing of important nutrients and vital substances, which in turn can trigger a chain reaction of problems, such as the contamination of body tissue, which then helps certain fungi and bacteria to move in.

An article in the German Doctors Newspaper described how a healthy intestinal flora improves overall health by reporting four out of five patients had normal and pain free bowel movements again. According to the article, this resounding success could be traced back to a preparation containing Escherichia coli or E. coli bacteria. In contrast to classic laxatives, bothersome flatulence and intestinal rumbling, abdominal cramps and nausea seldom appeared after the 8-week-long bacterial cure. Admittedly, there are still very few solid studies to indicate that probiotics (tablets containing live bacterial cultures) and prebiotics (nutrients which are supposed to stimulate certain good bacteria already found in the intestines) are of some use to health.

The primary objective should be to study exactly how certain foodstuffs, specific diets, drug consumption, toxins (pesticides, automobile exhaust, etc.), and stress effect the composition of the intestinal flora-and how this in turn influences human health (researchers are practically unanimous in that the intestinal flora influences health, but they continue to puzzle over how this
happens). But, evidently, this research work is neglected. Neither the EU (which financially facilitates studies of intestinal flora), nor the German Institute of Human Nutrition in Potsdam were willing to indicate to what extent they are active in this area. Instead the impression is given that here as well, the development of marketable products like functional food ingredients, specifically designed bacterial strains, or probiotics and prebiotics are the primary research targets.

This shows, once again, that the medical industry has little interest in real preventive research. The sale and application of antifungal preparations (just like antibiotics, antiviral medicines, vaccines, probiotics, etc.) makes a lot of money; the advice to eliminate, avoid, or reduce coffee, refined sugar or drugs, on the other hand, does not make any at all. And who really wants (or is able) to give up beloved habits? Many people would rather hope for a magic potion that makes all the aches and pains go away fast. Regretfully, this has led to the formation of a medical structure which ultimately only supports concepts that pass through the markets needle eye, and let’s company profits and experts salaries swell. The various hazards of this paradigm are shut out of the public conversation, and, so, we drift further and further from the possibilities of truly effective preventive health.

We must not ignore the fact that people are experiencing higher rates of fungal infections. It’s certainly not because fungi have become more aggressive, since they have hardly changed in the past millions of years. But what has changed is our behavior and with it our physical environment as well. We only have to glance at other areas of nature, where fungi can’t tell the difference between a human body and, for example, a forest. Everywhere, balance is at play: Excess substances are continuously generated, and must somehow be diminished again. If this were not the case, the earth would suffocate in the chaos of these excessively produced substances. This is where over 100,000 species of fungi come in and form their own kingdom next to animals and plants, acting like garbage collectors, eating up leaves, dead twigs, branches, tree stumps or pinecones in the forest, and bringing the nutrients back into the life cycle of the plants as re-utilizable humus.

Everything in nature-cells, our bodies, the land-occurs in a balance, which is why fungal illnesses in compact, healthy plants do not have a chance, as stated in botany textbook. Yet if a plant is infested by a fungus, then something must be wrong with the plants living conditions. This would be the case, for instance, if the plants soil were overly acidic, something which causes fungi to thrive.

For billions of years, nature has functioned as a whole with unsurpassed precision. Microbes, just like humans, are a part of this cosmological and ecological system. If humanity wants to live in harmony with technology and nature, we are bound to understand the supporting evolutionary principles ever better and to apply them properly to our own lives. Whenever we don’t do this, we create many ostensibly insolvable environmental and health problems of our time. These are thoughts which Rudolf Virchow (1821 -1902), a well-known doctor from Berlin, had when he required in 1875 that the doctor should never forget to interpret the patient as a whole being. The doctor will hardly understand the patient, then, if he or she does not see that person in the context of a larger environment.
Without the appearance of bacteria, human life would be inconceivable, as bacteria were right at the beginning of the development towards human life:

Progenotes (precursors to bacteria; ca. 3.5 billion years ago)
- Prokaryotes
- Anaerobic bacteria (anaerobe)
- Anaerobic photosynthetic bacteria
- Photosynthetic cyanobacteria
- Oxygen-rich atmosphere
- Aerobic breathing
- Aerobic prokaryotes
- Eukaryotes (1.6 - 2.1 billion years ago) Many-celled plants and animals
  - Mammals
  - Humans

With the term progenotes, bacteriologists denote a pre-preliminary stage, a life form from which prokaryotes (cells without nuclei) arise. Bacteria are known not to have cell nuclei, but they do have deoxyribonucleic acid (DNA) and ribonucleic acid (RNA), the carriers of genetic material. Anaerobic bacteria, as the word anaerobic indicates, can get by without oxygen. Only after the earth was supplied with oxygen could aerobic bacteria live; bacteria that formed the foundation for the lives of plants, animals, and humans.

Through this it becomes obvious that bacteria could very well exist without humans; humans, however, could not live without bacteria! It also becomes unimaginable that these mini-creatures, whose life-purpose and task for almost infinite time has been to build up life, are supposed to be the great primary or singular causes of disease and death. Yet, the prevailing allopathic medical philosophy has convinced us of this since the late 19th century, when Louis Pasteur and Robert Koch became heroes. Just a few hours after birth, all of a newborn baby’s mucous membrane has already been colonized by bacteria, which perform important protective functions. Without these colonies of billions of germs, the infant, just like the adult, could not survive. And, only an estimated one percent of our bacteria have even been discovered.

The majority of cells in the human body are anything but human: foreign bacteria have long had the upper hand, reported a research team from Imperial College in London under the leadership of Jeremy Nicholson in the journal Nature Biotechnology in 2004. In the human digestive tract alone, researchers came upon around 100 trillion microorganisms, which together have a weight of up to one kilogram. This means that the 1,000-plus known species of symbionts probably contain more than 100 times as many genes as exist in the host, as Nicholson states. It makes you wonder how much of the human body is human and how much is foreign?

Nicholson calls us human super-organisms—as our own ecosystems are reined by microorganisms. It is widely accepted, writes the Professor of Biochemistry, that most major disease classes have significant environmental and genetic components and that the incidence of disease in a population or individual is a complex product of the conditional probabilities of certain gene components interacting with a diverse range of environmental triggers. Above all,
nutrition has a significant influence on many diseases, in that it modulates complex communication between the 100 trillion microorganisms in the intestines! The microbes are part of our extended symbiotic genome and as such are in many ways just as important as our genes, says Nicholson.

How easily this bacterial balance can be decisively influenced can be seen with babies: if they are nursed with mother’s milk, their intestinal flora almost exclusively contains a certain bacterium (Lactobacillus bifidus), which is very different from the bacterium most prevalent when they are fed a diet including cow’s milk. The bacterium lactobacillus bifidus lends the breast-fed child a much stronger resistance to intestinal infections, for instance, writes microbiologist Dubos.

This is just one of countless examples of the positive interaction between bacteria and humans. But unfortunately, the knowledge that microorganisms can also do a lot of good for humans never enjoyed much popularity, Dubos points out. Humanity has made it a rule to take better care of the dangers that threaten life than to take interest in the biological powers upon which human existence is so decisively dependent. The history of war has always fascinated people more than descriptions of peaceful coexistence. And so it comes that no one has ever created a successful story out of the useful role that bacteria play in stomach and intestines. Alone the production of a large part of the food that lands on our plates is dependent on bacterial activity.

However, haven’t antibiotics helped or even saved the lives of many people? Without a doubt. But, we must note that as recently as 12 February 1941, the first patient was treated with an antibiotic, specifically penicillin. So, antibiotics have nothing to do with the increase in life expectancy, which really took hold in the middle of the 19th century (in industrialized countries), almost a century before the development of antibiotics. And, plenty of substances, including innumerable bacteria essential to life are destroyed through the administration of antibiotics, which directly translated from the Greek, means, against life. In the USA alone, millions of antibiotics are now unnecessarily administered. This has profound consequences, as antibiotics are held responsible for nearly a fifth of the more than 100,000 annual deaths that are traced back to side effects of medicines in the United States alone.

The over-use of antibiotics is also causing more bacteria to become resistant. Today, 70% of microbes held responsible for lung illnesses no longer respond to medications. The increase in resistance prompts the pharmaceutical sector to conduct more intensive research for new antibiotics. But the discovery of such molecules is a long, difficult and costly process (about $600 million per molecule). For many years, no important new antibiotic has come onto the market. At the same time, increasingly stronger preparations are being introduced, which only leads to the bacteria becoming even more resistant and excreting even more toxins.

A key question, such as the causes of pulmonary or middle-ear infection, cannot be answered by simply branding the microbes as lethal enemies and wiping them out. And yet people stick to vilifying the microbes because they are caught in their concept of the enemy and their tunnel vision is directed only at germs.

This is a perception that actually began with Louis Pasteur, who as an acclaimed researcher
spread the opinion that bacteria lingered everywhere in the air. And so Medicine Presents a Distorted Picture of Microbes the idea was born that bacteria (like fungi and viruses) would fatefully descend upon human and animal like swarms of locusts. For about ten years, doctors have speculated that even heart attacks are an infectious disease, triggered by the Chlamydia pneumonia bacterium. Because of this some patients were treated with antibiotics—but recently a study published in the New England Journal of Medicine stated quite plainly that there is no benefit from this.

Another issue when considering reports that E. coli bacteria have been detected in drinking water is the false notion that somehow on their forays these germs discovered a stream and then contaminated it. In fact, E. coli gets into drinking water through human or animal excrement, which serves as food for the bacteria.

Bacteria do not live isolated in an open atmosphere. Rather, they always exist together with cells and tissue parts. Just like a fungal culture, a bacterial culture does not simply consist of bacteria or fungi; rather, a terrain always exists as well. And depending on the (toxicity of a) terrain, there are different (toxic) germs. Let’s recall a well-known phrase from Claude Bernard (1813 - 1878), one of the best-known representatives of a holistic approach to health: The microbe is nothing, the terrain is everything.

If we ask bacteriologists which comes first: the terrain or the bacteria, the answer is always that it is the environment (the terrain) that allows the microbes to thrive. The germs, then, do not directly produce the disease. So, it is evident that the crisis produced by the body causes the bacteria to multiply by creating the proper conditions for actually harmless bacteria to mutate into poisonous pus-producing microorganisms.

Under close observation of disease progression, particularly in infective processes, damage to the organism occurs at the beginning of the disease—and only afterwards the bacterial activity begins, says general practitioner Johann Loibner. Everyone can observe this in himself. If we put dirt into a fresh wound, other bacteria appear as well. After the penetration of a foreign body, very specific germs appear which, after removal or release, go away on their own and do not continue to populate us. If we damage our respiratory mucous membrane through hypothermia, then those bacteria accordingly appear which, depending on the hypothermias acuteness and length, and the affected individual’s condition, can break down the affected cells and lead to expulsion, catarrh.

This would also explain what the dominant medical thought pattern can’t comprehend: why so many different microorganisms are in our bodies (among them such highly dangerous ones as the tuberculosis bacillus, the Streptococcus or the Staphylococcus bacterium) without bringing about any recognizable damage. They only become harmful when they have enough of the right kind of food. Depending on the type of bacterium this food could be toxins, metabolic end products, improperly digested food and much more.

Even surgery makes use of this principle, using little sacks of maggots to clean wounds that are particularly difficult to sanitize. The maggots eat only the dead or broken material. They do not touch healthy, living flesh. No surgeon in the world can cleanse such a wound so precisely and
safely as these maggots. And when everything is clean, the feast is over; the maggots don’t eat you up, because then they wouldn’t have anything more to eat.

Pasteur finally became aware of all of this, quoting Bernard’s dictum—the microbe is nothing, the terrain is everything—on his deathbed. But Paul Ehrlich (1854 - 1915), known as the father of chemotherapy, adhered to the interpretation that Robert Koch (just like Pasteur in his best days) preached: that microbes were the actual causes of disease. For this reason, Ehrlich, whom his competitors called Dr. Fantasy, dreamed of chemically aiming at bacteria, and decisively contributed to helping the magic bullets doctrine become accepted, by treating very specific illnesses successfully with very specific chemical-pharmaceutical preparations. This doctrine was a gold rush for the rising pharmaceutical industry with their wonder-pill production. But the promise of the magic bullet has never been fulfilled, writes Allan Brandt, a medical historian at Harvard Medical School.

This distorted understanding of bacteria and fungi and their functions in abnormal processes shaped attitudes toward viruses. At the end of the 19th century, as microbe theory rose to become the definitive medical teaching, no one could actually detect viruses. Viruses measure only 20 - 450 nanometers (billionths of a meter) across and are thus very much smaller than bacteria or fungi—so tiny, that one can only see them under an electron microscope. And the first electron microscope was not built until 1931. Bacteria and fungi, in contrast, can be observed through a simple light microscope. The first of these was constructed as early as the 17th century by Dutch researcher Antoni van Leeuwenhoek (1632 -1723).

Pasteurians were already using the expression virus in the 19th century, but this is ascribed to the Latin term virus (which just means poison) to describe organic structures that could not be classified as bacteria. It was a perfect fit with the concept of the enemy: if no bacteria can be found, then some other single cause must is responsible for the disease. In this case, a quote by Goethe's Mephistopheles comes to mind: For just where no ideas are, the proper word is never far.

The number of inconsistencies that arise from the theory of death-bringing viruses is illustrated by the smallpox epidemic, which even today people like to draw upon to stir up epidemic panic. But was smallpox really a viral epidemic that was successfully overpowered by vaccines? Medical historians doubt this, writes journalist Neil Miller in his book Vaccines: Are They Really Safe & Effective? Not only were there no vaccines for scarlet fever or the Black Plague, and these diseases disappeared all the same.

For example, in England, prior to the introduction of mandatory vaccinations in 1953, there were two smallpox deaths per 10,000 inhabitants per year. But at the beginning of the 1870s, nearly 20 years after the introduction of mandatory vaccinations, which had led to a 98% vaccination rate, England suffered 10 smallpox deaths per 10,000 inhabitants annually; five times as many as before. The smallpox epidemic reached its peak after vaccinations had been introduced, summarizes William Farr, who was responsible for compiling statistics in London.

From an orthodox view, the picture on the Philippines was no less contradictory: the islands experienced their worst smallpox epidemic at the beginning of the 20th century, even though the
vaccination rate was at almost 100%. And in 1928, a paper was finally published in the British Medical Journal that disclosed that the risk of dying from smallpox was five times higher for those who had been vaccinated than for those who had not.

In Germany statistics of smallpox mortalities have been collected since 1816. There were around 6,000 smallpox deaths per year until the end of the 1860s. In the years 1870 - 71, the number of victims suddenly jumped 14-fold to nearly 85,000 deaths. What had happened? The Franco-Prussian War was raging, and French prisoners of war were held in German camp under the most miserable conditions with extremely bad nutrition. As a result, the number of smallpox cases in the camps increased exponentially, even though all French and German soldiers had been vaccinated against smallpox. Germans (themselves suffering from the war) were likewise affected by the smallpox, although some of them had also been vaccinated. When the camps were dissolved directly after the war, the number of smallpox deaths also markedly declined.

Three years later, in 1874, there were only 3,345 smallpox deaths in Germany per year. Prevailing medicine says that this reduction was due to the Reichsimpfgesetz, a law that among other things stipulated that a child had to be vaccinated before the end of the calendar year following his year of birth. But in fact, this law first came into effect in 1875, when the smallpox scare was long past. Improvements in hygiene, technology, and civilization much had occurred at that time, which led to the reduction in illnesses and deaths, says physician Gerhard Buchwald. Irrespective of this, mainstream viral research and medicine exclusively assumes that viruses are infectious pathogenic germs, which actively spread out in the cells in a parasitic way (with the assistance of enzymes and other cellular components) and multiply-ultimately attacking and sometimes killing cells. Or as a well-known German daily newspaper puts it, in the typical sensationalized manner: Viruses are the earth’s wiliest infectious agents: they attack animals and humans to enslave their cells.

As thrilling as this may sound, no scientific backing is provided for this statement. To accept this, the existence of these so-called killer viruses must first be proven. And this is where the trouble begins. Consequential, scientifically-sound evidence has never been provided, even though it’s as easy as taking a sample of patient blood and isolating one of these viruses, in a purified form with its complete genetic material (genome) and virus shell, directly from it, and then imaging it with an electron microscope. But these critical initial steps have never been done with HSN l (avian flu), the so-called hepatitis C virus, HIV, and numerous other particles that are officially called viruses and depicted as attack-crazy beasts.

At this point, we encourage our readers to verify dominant virus theories independently—as many people have done, among them Nobel laureates, top microbiologists and researchers from other fields, serious journalists and lay people alike. We’ve asked for evidence from important institutions like World Health Organization (WHO), the American Centers for Disease Control (CDC), or its German counterpart, the Robert Koch Institute (RKI) in Berlin. In the summer of 2005, for example, we contacted the RKI and requested the following information:

1. Please name the studies that indisputably show that the SARS, hepatitis C, Ebola, smallpox and polio viruses and the BSE causative agent have been proven to exist (complete purification, isolation and definition of biochemical properties plus electron micrographs).
2. Please name studies that indisputably show that the viruses named above cause disease (and also that other factors like malnutrition, toxins, etc. do not at least co-determine the course of disease).
3. Please name at least two studies that indisputably show that vaccinations are effective and active.

Unfortunately, to date we have not (despite repeated questioning) yet had a single study named to us. Readers may wonder how it can be continually claimed that this or that virus exists and has potential to trigger diseases through contagion. An important aspect in this context is that some time ago, mainstream virus-science left the road of direct observation of nature, and decided instead to go with so-called indirect proof with procedures such as antibody and PCR tests. In this book, we will often stray from the well-traveled road, but at this point we must point out that these methods lead to results which have little to no meaning.

Antibody tests just prove the existence of antibodies-and not the virus or particle itself to which the antibody tests react. That means: as long as the virus or cell particle (antigen) has not been precisely defined, no one can say what these antibody tests are reacting to; they are thus unspecific in medical lingo.

It is no different with PCR (polymerase chain reaction), which is used to track down genetic sequences, little genetic snippets, and then replicate them a million fold. As with antibody tests, PCR probably has significance because it displays a sort of immune reaction (as it is called in technical terms) in the body; or, to put it more neutrally, some sort of disturbance or activity on a cellular level. But a virus with indeterminate characteristics cannot be proven by PCR any more than it can be determined by a little antibody test. Again, this is because the exact virus determination has not been carried out.

In terms of genetics, these short pieces that are found using the PCR are not complete and do not even satisfy the definition of a gene (of which humans are said to have 20,000 to 25,000). In spite of this, it is suggested that pasted together they would depict the whole genetic material of a given virus. But nobody has presented a paper that shows an electron micrograph of this so-called reproduced virus.

Even if scientists assume that the particles discovered in the laboratory (antigens and gene snippets) are the viruses mentioned, this is a long way from proving that the viruses are the causes of the diseases in question, particularly when the patients or animals that have been tested are not even sick, which, often enough is the case. Another important question must be raised: even when a supposed virus does kill cells in the test-tube (in vitro), or lets embryos in a chicken egg culture die, can we safely conclude that these findings can be carried over to a living organism (in vivo)? Many issues contradict this theory, such as that the particles termed viruses stem from cell cultures (in vitro) whose particles could be genetically degenerate because they have been bombarded with chemical additives like growth factors or strongly oxidizing substances.

In 1995, the German news magazine Der Spiegel delved into this problem (something that is worth noting, when one considers that this news magazine usually runs only orthodox virus
coverage), quoting researcher Martin Markowitz from the Aaron Diamond AIDS Research Center in New York: The scientist [Markovitz] mauls his virus-infected cell cultures with these poisons in all conceivable combinations to test which of them kill the virus off most effectively. Of course, we don’t know how far these cross-checks in a test-tube will bring us, says Markowitz.

What ultimately counts is the patient. His clinical experience has taught him the difference between test-tube and sick bed. He is more aware than most AIDS researchers of how little the behavior of cultured virus stems in incubator cells has to do with those that grow naturally in a network of hormones, antibodies, scavenger and T cells of the immune system of a living person. Andreas Meyerhans, from the Institute Pasteur in Paris uses the phrase: To culture is to disturb, which basically means that the results obtained in vitro only confuse.

Unfortunately, the decade is characterized by climbing death rates, caused by lung cancer, heart disease, traffic accidents and the indirect consequences of alcoholism and drug addiction, wrote Sir Frank Macfarlane Burnet, recipient of the Nobel Prize for Medicine, in his 1971 book Genes Dreams, and Realities: The real challenge of the present day is to find remedies for these diseases of civilization. But nothing that comes out of the labs seems to be significant in this context; laboratory research’s contribution has practically come to an end. For someone who is well on the way to a career as a lab researcher in infectious disease and immunology, these are not comforting words.

To biomedical scientists and the readers of their papers, Burnet continued, it may be exciting to hold forth on the detail of a chemical structure from a phages [viruses from simple organisms; see below] RNA, or the production of antibody tests, which are typical of today’s biological research. But modern fundamental research in medicine hardly has a direct significance to the prevention of disease or the improvement of medical precautions.

But mainstream medicine avoids this theory like the devil does holy water. Instead, one tries to demonstrate the pathogenicity (ability to cause disease) of these particles through experiments that could hardly be more arcane. For instance, test substrates were injected directly into the brains of lab animals. This was the procedure with BSE and polio, for example; and even the famous Louis Pasteur had applied this method in his rabies experiments, in which he injected diseased brain tissue into the heads of dogs (Pasteur became famous through these experiments, and only years after his death were these studies found to be pure put-on). The industry now says that direct injections into the brain are unrealistic, and thus ultimately provide no evidence of pathogenic effects.

Why not suppose that a virus, or what we term a virus, is a symptom—i.e. a result—of a disease? Medical teaching is entrenched in Pasteur and Kochs picture of the enemy, and has neglected to pursue the thought that the body’s cells could produce a virus on its own accord, for instance as a reaction to stress factors. The experts discovered this a long time ago, and speak of endogenous viruses—particles that form inside the body by the cells themselves. In this context, the research work of geneticist Barbara McClintock is a milestone.

In her Nobel Prize paper from 1983, she reports that the genetic material of living beings can
constantly alter, by being hit by shocks. These shocks can be toxins, but also other materials that produced stress in the test-tube. This in turn can lead to the formation of new genetic sequences, which were unverifiable (in vivo and in vitro) before.

Long ago, scientists observed that toxins in the body could produce physiological reactions, yet current medicine sees this only from the perspective of exogenous viruses. In 1954, the scientist Ralph Scobey reported in the journal Archives of Pediatrics, that herpes simplex had developed after the injection of vaccines, the drinking of milk or the ingestion of certain foodstuffs; while herpes zoster (shingles) arose after ingestion or injection of heavy metals like arsenic and bismuth or alcohol.

It is also conceivable that toxic drugs like poppers, recreational drugs commonly used by homosexuals, or immunosuppressive medications like antibiotics and anti-virals could trigger what is called oxidative stress. This means that the blood's ability to transport oxygen, so important for the life and survival of cells, is compromised. Simultaneously, nitric oxides are produced, which can severely damage cells. As a result, antibody production is stirred up, which in turn causes the antibody tests to come out positive. Also, new genetic sequences are generated through this, which are then picked up by the PCR tests—all this, mind you, without a pathogenic virus that attacks from outside.

But prevailing medicine condemns such thoughts as heresy. Just as the orthodoxy fought against McClintock’s concept of jumping genes for decades, because they did not want to let go of their own model of a completely stable genetic framework. Here, they had not merely ignored McClintock, but even became downright hostile, according to McClintock. Looking back, it is painful to see how extremely fixated many scientists are on the dominant assumptions, on which they have tacitly agreed, McClintock wrote in 1973, shortly after the medical establishment admitted, finally, that she had been right. One simply has to wait for the right time for a change in conception. However, McClintock had no time to brace herself against the prevailing HN = AIDS dogma. She did voice criticism that it has never been proven AIDS is triggered by a contagious virus. But the Nobel Prize winner died in 1992, shortly after increased numbers of critics of the HN = AIDS dogma had come into the game.

Whether Nobel laureate or layperson, ask yourself this simple question: how is it actually imaginable that killer viruses stalk the world bumping off one human cell after another? Viruses—as opposed to bacteria and fungi—do not even have their own metabolisms. By definition, viruses have completely given their metabolisms to the cells. They are composed of only one nucleic acid strand (DNA or RNA ·genes) and one protein capsule, so are missing the decisive attributes of living beings. Strictly speaking, they do not count among microbes, which comes from the Greek: micro = small, bios = life. How can viruses, like bacteria, be in a position to become active and aggressive of their own accord? Remember, it is said that viruses may have existed for three billion years. And exactly like bacteria and fungi, viruses are also said to be ubiquitous from the deep sea to the polar ice caps. A 2006 study published in the Proceedings of the National Academy of Sciences found that there are more than 20,000 species of bacteria in a liter of seawater—the researchers had expected to find only 1,000 to 3,000 species.

Just as scientists have discovered through ever more powerful telescopes that stars number in the
billions, we are learning that the number of marine organisms invisible to the eye exceeds all expectations and their diversity is much greater than we could have imagined, says lead author Mitchell Sogin, director of the Massachusetts-based Marine Biological Laboratory (MBL) Center for Comparative and Molecular Biology and Evolution. This study shows we have barely scratched the surface. The number of different kinds of bacteria in the oceans could eclipse five to 10 million. Furthermore, one liter of sea water is said to contain no less than 10 billion viruses of very simple organisms, like single-celled algae, called (bacterio) phages; umpteen times as many viruses (phages) as bacteria. Both of these discoveries—the long development time and their universal existence—argue clearly that nature, which constantly strives for balance, lives in symbiosis with these viruses.

Luckily, the phages omnipresence has flown below the radar of prevailing medical viral research—otherwise there would probably be regulations against bathing in the sea without full-body condoms or epidemic-protection suits, and only under the condition that we first take prophylactic antiviral medications. Or, why not try to disinfect large surfaces of seawater. We are already well on the way to this kind of thinking, since phages are already being presented as super villains that work using wily tricks. But there is no real proof here either.

We’d be wise to remember times in which the ruling dogma of viral killers was (freely and openly) sharply attacked and dismissed as pure belief. Indeed, there were many prominent microbiologists who insisted that bacteriophages just aren’t viruses, but rather products endogenously produced, i.e. by bacteria. Robert Doerr, editor of the Handbook of Virology, published by Springer in 1938, even held the idea that not only phages, but also other viruses were the product of cells. Let’s look at one of their arguments: bacteriophages cannot be living entities that become active independently, since phages themselves cannot be destroyed by temperatures as high as 120 degrees. And it would probably be of use to recall the history of this decade-long dispute, says Dutch microbiologist Ton van Helvoort, for controversies and finding consensus are at the heart of scientific research.

Chapter 2
The elevated status Louis Pasteur enjoyed during his lifetime is made clear by a quotation from physician Auguste Lutaud in 1887 (eight years before Pasteur’s death): In France, one can be an anarchist, a communist or a nihilist, but not an anti-Pasteurian. In truth, however, Pasteur was no paragon with a divinely pure clean slate, but rather a researcher addicted to fame acting on false assumptions and he misled the world and his fellow scientists about the research behind two of his most famous experiments, as the journal The Lancet stated in 2004.

In his downright fanatical hate of microbes, Pasteur actually came from the ludicrous equation that healthy (tissue) equals a sterile (germ-free) environment. He believed in all earnestness that bacteria could not be found in a healthy body/ and that microbes flying through the air on dust particles were responsible for all possible diseases. At 45 years of age, he was basking in his fame, as bacteriologist Paul de Kruif writes in his book Microbe Hunters, and trumpeted his hopes out into the world: it must lie within human power to eliminate all diseases caused by parasites [microbes] from the face of the earth.

Flaws in Pasteur’s theories were shown long ago in the first half of the 20th century by
experiments in which animals were kept completely germ-free. Their birth even took place by Cesarean section; after that, they were locked in microbe free cages and given sterile food and water—after a few days, all the animals were dead. This made it apparent that contamination by exogenous bacteria is absolutely essential to their lives.

In the early 1960s, scientists succeeded for the first time in keeping germ-free mice alive for more than a few days, namely for several weeks. Seminal research on these germ-free rodents was performed by Morris Pollard in Notre-Dame, Indiana. However, this does not undermine the fact that germs are essential for life. Not only do mice under natural conditions have a life span of three years, which is much longer than the average life span of these germ-free lab animals. The ability to keep germ-free animals such as mice or rats alive for a longer time requires highly artificial lab conditions in which the animals are synthetically fed with vitamin supplements and extra calories, conditions that have nothing to do with nature.

These specially designed liquid diets are needed because under normal rearing conditions, animals harbor populations of microorganisms in the digestive tract. These microorganisms generate various organic constituents as products or byproducts of metabolism, including various water-soluble vitamins and amino acids.

In the rat and mouse, most of the microbial activity is in the colon, and many of the microbially produced nutrients are not available in germ-free animals. This alters microbial nutrient synthesis and, thereby, influence dietary requirements. Adjustments in nutrient concentrations, the kinds of ingredients, and methods of preparation must be considered when formulating diets for laboratory animals reared in germ-free environments or environments free of specific microbes. One important target by administering these artificial diets is to avoid the accumulation of metabolic products in the large intestine. However, it has been observed that already after a short time the appendix or cecum of these germ-free reared rodents increased in weight and eventually became abnormally enlarged, filled with mucus which would normally have been broken down by microbes.

Furthermore, in germ-free conditions rodents typically die of kidney failure a sign that the kidneys are overworked in their function as an excretion organ if the large intestine has been artificially crippled. In any case, it shows that germ-free mice would not be able to survive and reproduce while staying healthy in realistic conditions, which can never be duplicated by researchers, not even approximately.

Apart from this, it is not clear that these germ-free animals have been truly 100% germ-free. Obviously not all tissues and certainly not every single cell could have been checked for germs. Nobody can know that these animals are absolutely germfree, especially if one keeps in mind that germs such as the Chlamydia trachomatis may hide so deeply in the cells that they persist there even after treatment with penicillin.

Furthermore, even if the specimens of so-called germ-free animals are maintained under optimum conditions—assumed to be perfectly sterile—their tissues do, nevertheless, decay after a time, forming spontaneous bacteria. But how do we explain these spontaneous bacteria? They cannot come from nothing, so logic allows only one conclusion: the bacteria must have already
been present in the so-called germ-free mice (in any case, mice said to be bacteria-free are apparently not virus-free; this was demonstrated in 1964 in the Journal of Experimental Medicine by Etienne de Harven who observed, by electron microscopy, typical so-called retroviral particles in the thymus of germ-free Swiss and C3H mice; of course, these viruses may be endogenous retroviruses which sometimes are expressed as particles-but of endogenous origin).

If nature wanted us bacteria-free, nature would have created us bacteria-free. Germ-free animals, which apparently aren’t really germ-free, can only exist under artificial lab conditions, not in nature. The ecosystems of animals living under natural conditions-be it rodents or be it human beings-depend heavily upon the activities of bacteria, and this arrangement must have a purpose. But back to Tricky Louis who deliberately lied, even in his vaccination experiments, which provided him a seat on the Mount Olympus of research gods. In 1881, Pasteur asserted that he had successfully vaccinated sheep against anthrax.

But not only does nobody know how Pasteur’s open land tests outside the Paris gates really proceeded, but the national hero of Ia grande Nation, as he would later be called, had in fact clandestinely lifted the vaccine mixture from fellow researcher Jean-Joseph Toussaint, whose career he had earlier ruined through public verbal attacks. And what about Pasteur’s purportedly highly successful experiments with a rabies vaccine in 1885? Only much later did the research community learn that they did not satisfy scientific standards at all, and were thus unfit to back up the chorus of praise for his vaccine-mixture? Pasteur’s super-vaccine might have caused rather than prevented rabies, writes scientific historian Horace Judson.

These experiments weren’t debated for decades largely due to the fastidious secretiveness of the famous Frenchman. During his lifetime, Pasteur permitted absolutely no one-not even his closest co-workers-to inspect his notes. And Tricky Louis arranged with his family that the books should also remain closed to all even after his death. In the late 20th century, Gerald Geison, medical historian at Princeton University, was first given the opportunity to go through Pasteur’s records meticulously, and he made the fraud public in 1995. That it became so controversial shouldn’t be particularly surprising, for sound science thrives in a transparent environment so that other researchers can verify the conclusions made.

Secretiveness has an oppositional goal: shutting out independent monitoring and verification. When external inspection and verification by independent experts are shut out of the process, the floodgates are open to fraud. Of course, we observe this lack of transparency everywhere, be it in politics, in organizations like the international Football association FIFA, and also in scientific communities [that] believe that public funding is their right, but so is freedom from public control, according to Judson. With this, mainstream research has actually managed to seal off their scientific buildings from public scrutiny.

This set-up lacks critical checks and balances, so no one is ultimately in the position to scrutinize the work of researchers and make sure research is conducted in an honest way. We are left to simply trust that they go about it truthfully. But a survey taken by scientists and published in a 2005 issue of Nature showed that a third of researchers admitted they would not avoid deceptive activities, and would simply brush to the side, any data that did not suit their purposes. A crucial
aspect of science has been lost; few researchers now trouble themselves to verify data and conclusions presented by fellow researchers.

Such quality checkups are equated with a waste of time and money and for that reason are also not financed. Instead medical researchers are completely occupied obsessed with chasing after the next big high-profit discovery. And many of today’s experiments are constructed in such a complicated manner that they cannot be reconstructed and precisely verified at all. This makes it very easy for researchers to ask themselves, without having to fear any consequences: why shouldn’t I cheat?

One would hope that the so-called peer review system largely eliminates fraud. It is still commonly considered a holy pillar of the temple of science, promising adherence to quality standards. But the decades-long practice of peer review is rotten to the core. It functions like this: experts (peers) who remain anonymous examine (review) research proposals and journal articles submitted by their scientific competitors. These so-called experts then decide if the proposals should be approved or the articles printed in scientific publications. There are said to be around 50,000 such peer reviewed publications, and all the best known journals such as Nature, Science, New England Journal of Medicine, British Medical Journal and The Lancet, are peer reviewed. There is, however, a fundamental problem: peer reviewing, in its current form, is dangerously flawed. If researchers in other fields conducted studies and published results using this process, what would happen? If their current methods were common in the car industry, for example, BMWs competitors could decide, through an anonymous process, whether or not BMW would be permitted to develop a new car model and bring it to the market. Clearly this would stifle innovation and invite conflicts of interest and fraud.

Peer review is slow, expensive, a profligate of academic time, highly selective, prone to bias, easily abused, poor at detecting gross defects, and almost useless for detecting fraud, says Richard Smith, former Editor in Chief of the British Medical Journal. No wonder, then, that all the cases of fraud which scientific historian Judson outlines in his 2004 book The Great Betrayal: Fraud in Science were not uncovered by the peer review system, but rather by pure coincidence. And next to Pasteur in the pantheon of scientific fraudsters appear such illustrious names as Sigmund Freud and David Baltimore, one of the best-known recipients of the Nobel Prize for medicine (well discuss Baltimore in more detail later in this chapter).

The other shining light of modern medicine, German doctor Robert Koch (1843 - 1910) was also an enterprising swindler. At the 10th International Medical Congress in Berlin in 1890, the microbe hunter with the oversized ego pronounced that he had developed a miracle substance against tuberculosis. And in the German Weekly Medical Journal Koch even claimed his tests on guinea pigs had proved that it was possible to bring the disease completely to a halt without damaging the body in other ways.

The reaction of the world-at-large to this alleged miracle drug Tuberkulin was at first so overwhelming that in Berlin, Kochs domain, sanatoria shot out of the ground like mushrooms. Sick people from all over the world turned the German capital into a son of pilgrimage site.
But soon enough, Tuberkulin was found to be a catastrophic failure. Long-term cures did not emerge, and instead one hearse after another drove up to the sanatoria. And newspapers such as the New Year’s edition of the satirical Der wahre Jakob (The Real McCoy) jeered: Herr Professor Koch! Would you like to reveal a remedy for dizziness bacteria!

In the style of Pasteur, Koch had also kept the contents of his alleged miracle substance strictly confidential at first. But as death rates soared, a closer inspection of the drugs properties revealed that Tuberkulin was nothing more than a bacillus culture killed off by heat; even with the best of intentions, no one could have assumed that it would have helped tuberculosis patients suffering from severe illness. On the contrary, all individuals—be it the test patients or the ones who were given it later as an alleged cure—experienced dramatic adverse reactions: chills, high fever, or death.

Finally, Koch’s critics, including another medical authority of that time, Rudolf Virchow, succeeded in proving that Tuberkulin could not stop tuberculosis. Rather, it was feared, according to the later scathing criticisms, that it made the diseases progress even worse. Authorities demanded that Koch brings forth evidence for his famous guinea pig tests—but he could not.

Experts such as historian Christoph Gradmann of Heidelberg say that Koch cleverly staged Tuberkulins launch. Everything seemed to have been planned well in advance. In late October 1890, during the first wave of Tuberkulin euphoria, Koch had taken leave of his hygiene professorship. In confidential letters, he requested his own institute-modeled on the Institute Pasteur in Paris—from the Prussian state in order to be able to research his Tuberkulin extensively. Professor Koch calculated the expected profit on the basis of a daily production of 500 portions of Tuberkulin at 4.5 million marks annually. On the reliability of his prognosis, he dryly observed: Out of a million people, one can reckon, on average, with 6,000 to 8,000 who suffer from pulmonary tuberculosis. In a country with a population of 30 million, then, there are at least 180,000 phthisics (tubercular people). Kochs announcement in the German Weekly Medical Journal appeared simultaneously with excessively positive field reports by his confidantes, according to Gradmann, served for the verification of Tuberkulin just as much as for its propaganda.

At the end of the 19th century, when Pasteur and Koch became celebrities, the general public had hardly a chance to brace itself against microbe propaganda. Medical authorities, who adhered to the microbes = lethal enemies theory, and the rising pharmaceutical industry already had the reins of power and public opinion firmly in their hands. With this, the course was set for the establishment of clinical studies using laboratory animals, with the goal of developing (alleged) miracle pills against very specific diseases.

The scheme was so effective that even a substance like Tuberkulin, which caused such a fatal disaster, was highly profitable. Koch never even admitted that his Tuberkulin had been a failure. And Hoechst, a dye factory looking for a cheap entry into pharmaceutical research, got into Tuberkulin manufacturing. Kochs student Arnold Libbertz was to supervise production, with
close cooperation from Kochs institute, and the rising pharmaceutical industry were decisively spurred on.

From this point on, scientists tried to squeeze virtually everything into the model one disease-one cause (pathogen)-one miracle cure, something that prompted one failure after another. For example, for a long time, the prevailing medicine spiritedly asserted that diseases like scurvy (seamen’s disease), pellagra (rough skin), or beriberi (miners and prisoners disease) were caused by germs. Until the orthodoxy ultimately, with gritted teeth, admitted that vitamin deficiency is the true cause.

With beriberi, for instance, it was decades before the dispute over what caused the degenerative neural disease took its decisive turn when vitamin B1 (thiamine) was isolated in 1911—a vitamin that was absent in refined foods like white rice. Robert R. Williams, one of the discoverers of thiamine, noted that, through the work of Koch and Pasteur, all young physicians were so imbued with the idea of infection as the cause of disease that it presently came to be accepted as almost axiomatic that disease could have no other cause [than microbes]. The preoccupation of physicians with infection as a cause of disease was doubtless responsible for many digressions from attention to food as the causal factor of beriberi.

The idea that certain microbes—above all fungi, bacteria and viruses—are our great opponents in battle, causing certain diseases that must be fought with special chemical bombs, has buried itself deep into the collective conscience. But a dig through history reveals that the Western world has only been dominated by the medical dogma of one disease, one cause, one miracle pill since the end of the 19th century, with the emergence of the pharmaceutical industry. Prior to that, we had a very different mindset, and even today, there are still traces everywhere of this different consciousness.

Since the time of the ancient Greeks, people did not catch a disease, they slipped into it. To catch something meant that there was something to catch, and until the germ theory of disease became accepted, there was nothing to catch, writes previously mentioned biology professor Edward Golub in his work, The Limits of Medicine: How Science Shapes Our Hope for the Cure. Hippocrates, who is said to have lived around 400 B.C., and Galen (one of the most significant physicians of his day; born in 130 A.D.), represented the view that an individual was, for the most part, in the driver’s seat in terms of maintaining health with appropriate behavior and lifestyle choices.

Most disease [according to ancient philosophy] was due to deviation from a good life, says Golub. [And when diseases occur] they could most often be set aright by changes in diet—[which] shows dramatically how 1,500 years after Hippocrates and 950 years after Galen, the concepts of health and disease, and the medicines of Europe, had not changed far into the 1911 century. Even into the 1850s, the idea that diseases are contagious found hardly any support in medical and scientific circles. One of the most significant medical authorities of the time was the German Max von Pettenkofer (1818-1901), who tried to comprehend things as wholes, and so incorporated various factors into his considerations about the onset of diseases, including individual behavior and social conditions. To von Pettenkofer, the microbe-theoreticians
oversimplified, monocausal hypothesis seemed naive, something that turned him into a proper anti-contagionist.

In view of the then-emerging division of medicine into many separate specialized disciplines, the scientist, later appointed rector of the University of Munich, jeered: Bacteriologists are people who don’t look further than their steam boilers, incubators and microscopes.

And so it was also von Pettenkofer who at this time directed the discussion on the treatment of cholera, a disease so typical to rising industrial nations in the 19th century. He held the same position that the famous doctor François Magendie (1783 - 1855) had adopted back in 1831, when he reported to the French Academy of Sciences that cholera was not imported, nor contagious, but rather it was caused by excessive dirt as a result of catastrophic living conditions. Correspondingly, the poorest quarters in centers like London were, as a rule, also the ones most afflicted by cholera.

Von Pettenkofer identified drinking water as the main cause. There were no treatment plants in those days, so water was often so visibly and severely contaminated with industrial chemicals and human excrement that people regularly complained about its stink and discoloration. Studies also showed that households with access to clean water had few to no cholera cases at all. Although von Pettenkofer certainly didn’t deny the presence of microbes in this cesspool, he argued that these organisms could contribute to the diseases course, but only when the biological terrain was primed so they could thrive.

Unfortunately, von Pettenkofer’s authority ultimately could not prevent adherents of the microbe theory from taking the matter into their own hands at the end of the 19th century, and they squeezed cholera into their narrow explanatory concept as well. So a microbe (in this case the bacterium Vibrio cholerae or its excretions) was branded as the sole culprit—and Pasteurian microbe theory was falsely decorated for having repelled cholera. Golub was left shouting into the void: Why does Pasteur get the credit for that which the sanitation movement and public health were primarily responsible?

The 1500-year history of a holistic view of health and disease was much too connected with life and its monstrous complexities to disappear altogether at the spur of the moment. Yet, it virtually disappeared from the collective conscience. Geneticist Barbara McClintock was of the opinion that the concepts that have since posed as sound science cannot sufficiently describe the enormous multi-layered complexities of all forms of natural life, and with that, their secrets. Organisms, according to the Nobel Prize winner for medicine, lead their own lives and comply with an order that can only be partially fathomed by science. No model that we conceive of can even rudimentarily do justice to these organisms incredible capability to find ways and means of securing their own survival.

By the beginning of the 1970s, Nobel laureate for medicine, Sir Frank Macfarlane Burnet had also become very skeptical about the usefulness of molecular biology, [especially because of] the impossible complexity of living structure and particularly of the informational machinery of the cell. [Certainly, molecular biologists are] rightly proud of their achievements and equally rightly feel that they have won the right to go on with their research. But their money comes from
politicians, bankers, foundations, who are not capable of recognizing the nature of a scientist’s attitude to science and who still feel, as I felt myself 30 years ago, that medical research is concerned only in preventing or curing human disease. So our scientists say what is expected of them, their grants are renewed and both sides are uneasily aware that it has all been a dishonest piece of play-acting—but then most public functions are.

Certainly not all doctors have clamored for roles on the medical industrial stage and some were key players in keeping the holistic health viewpoint alive. Swiss doctor Maximilian Bircher-Benner (1867 - 1939) directed his attention to the advantages of nutrition after treating his own jaundice with a raw foods diet, as well as a patient suffering from severe gastric problems. In 1891, long before the significance of vitamins and dietary fiber to the human body had been recognized, Bircher-Benner took over a small city practice in Zurich, where he developed his nutritional therapy based on a raw foods diet.

By 1897, only a few years later, the practice had grown into a small private clinic, where he also treated in patients. There was strong interest in his vegetarian raw food diet from all over the world, so, Bircher-Benner erected a four-story private sanatorium in 1904 called Lebendige Kraft (living force). And so besides a raw foods diet, Bircher-Benner (whose name has been immortalized in Bircher-Muesli) promoted natural healing factors like sun-baths, pure water, exercise and psychological health. With this, he supported treatments that had become increasingly neglected with the appearance of machines and, particularly, pharmaceuticals: attention to the natural healing powers of the body and the body’s cells, which possess their own sort of sensitivity and intelligence.

Walter Cannon, professor of physiology at Harvard, also made holistic health his central theme, in his 1932 work The Wisdom of the Body. Here, he describes the concept of homeostasis, and underlines that occurrences in the body are connected with each other and self-regulating in an extremely complex way. 64 Wisdom of the Body is an attribute of living organisms, wrote Israeli medical researcher Gershom Zajicek in a 1999 issue of the journal Medical Hypotheses. It directs growing plants toward sunshine, guides amoebas away from noxious agents, and determines the behavior of higher animals. The main task of the wisdom of the body is to maintain health, and improve its quality. The wisdom of the body has its own language and should be considered when examining patients.

The words of biologist Gregory Bateson from 1970 are certainly still valid today: [Walter] Cannon wrote a book on the Wisdom of the Body; but nobody has written a book on the wisdom of medical science, because that is precisely the thing it lacks.

After World War II, diseases such as tuberculosis, measles, diphtheria or pneumonia no longer triggered mass fatalities in industrialized nations such as affluent America. This became a huge problem for institutions like the Centers for Disease Control (CDC), the American epidemic authorities, as redundancy threatened. In 1949, a majority voted to eliminate the CDC completely. Instead of bowing out of a potentially very lucrative industry, the CDC went on an arduous search for viruses. But, how to find an epidemic where there isn’t any? You do clustering.
This involves a quick scan of your environment—hospitals, daycares, local bars, etc.—to locate one, two, or a few individuals with the same or similar symptoms. This is apparently completely sufficient for virus hunters to declare an impending epidemic. It doesn’t matter if these individuals have never had contact with each other, or even that they’ve been ill at intervals of weeks or even months. So, clusters can deliver no key clues or provide actual proof of an existing or imminent microbial epidemic.

Even the fact that a few individuals present the same clinical picture does not necessarily mean that a virus is at work. It can mean all sorts of things including that afflicted individuals had the same unhealthy diet or that they had to fight against the same unhealthy environmental conditions (chemical toxins etc.). Even an assumption that an infectious germ is at work could indicate that certain groups of people are susceptible to a certain ailment, while many other people who are likewise exposed to the microbe remain healthy.

For this reason, epidemics rarely occur in affluent societies, because these societies offer conditions (sufficient nutrition, clean drinking water, etc.) which allow many people to keep their immune systems so fit that microbes simply do not have a chance to multiply abnormally (although antibiotics are also massively deployed against bacteria; and people who overuse antibiotics and other drugs that affect the immune system are even at greater risk).

Just how ineffective clustering is in finding epidemics becomes evident, moreover, if we look more closely at cases where clustering has been used as a tool to sniff out (allegedly impending) epidemics. This happened with the search for the causes of scurvy, beriberi and pellagra at the beginning of the 20th century. But, as illustrated, it proved groundless to assume that these are infectious diseases with epidemic potential.

The best-known example in recent times is HIV/AIDS. At the beginning of the 1980s, a few doctors tried to construct a purely viral epidemic out of a few patients who had cultivated a drug-taking lifestyle that destroyed the immune system. Well discuss how virus authorities manufactured this epidemic in Chapter 3. For now, well quote CDC officer Bruce Evatt, who admitted that, the CDC went to the public with statements for which there was almost no evidence. We did not have proof it was a contagious agent.

Unfortunately, the world ignored all kinds of statements like this. So talk of the AIDS virus has since kept the world in epidemic fear and virus hunters are now the masters of the medical arena. Every cold, every seasonal influenza, hepatitis disease, or whatever other syndrome has become an inexhaustible source for epidemic hunters armed with their clustering methods to declare ever new epidemics that pose threats to the world.

In 1995, allegedly, the microbe from hell came to England, according to media scientist Michael Tracey, who was then active in Great Britain and collected media headlines like, Killer Bug Ate My Face, Flesh Bug Ate My Brother in 18 Hours and Flesh Eating Bug Killed My Mother in 20 Minutes. Tracey writes, The Star was particularly subtle in its subsidiary headline, it starts with a sore throat but you can die within 24 hours. Yet the bacterium, known to the medical world as Streptococcus A, was anything but new. Usually only a few people die from it each year, says Tracey. In that year in England and Wales just 11 people. The chances of getting infected were
infinitesimally small but that didn’t bother the media an all. A classic example of bad journalism triggering a panic.

In the same year, the US CDC sounded the alarm, warning insistently of an imminent Ebola virus pandemic. With the assistance of cluster methods, several fever cases in Kikwit, in the Democratic Republic of Congo, were separated out and declared as an outbreak of the Ebola epidemic. In their addiction to sensation the media reported worldwide that a deadly killer virus was about to leave its jungle lair and descend on Europe and the USA.

Time magazine showed spectacular pictures of CDC detectives in spacesuits impermeable to germs and colorful photographs in which the dangerous pathogen could ostensibly be seen. The director of the UN AIDS program made the horror tangible by imagining: It is theoretically possible that an infected person from Kikwit makes it to the capital, Kinshasa, climbs into a plane to New York, gets sick and then poses a risk to the USA. Within a month, however, Ebola was no longer a problem in Africa, and not one single case was ever reported in Europe or North America’s. And a publication in which the Ebola virus is characterized (with its genetic material and virus shell) and shown in an electron micrograph is still nowhere to be found.

Practically all of the infectious illnesses that infected people in industrialized countries in the decades before World War II (tuberculosis etc.) ceased to cause problems after 1945. For a few years, the major exception was polio (infantile paralysis), which continues to be called an infectious disease. In the 1950s, the number of polio cases in developed countries fell drastically- and epidemic authorities attributed this success to their vaccination campaigns. But a look at the statistics reveals that the number of polio victims had already fallen drastically when vaccination activities started.

Many pieces of evidence justify the suspicion that the cause of infantile paralysis (polio) is not a virus. Many experts, like American physician Benjamin Sandler, believe a decisive factor is a high consumption of refined foods such as granulated sugar. Others cite mass vaccinations. Indeed, since the beginning of the 20th century, it has been known that the paralysis so typical of polio have often appeared at the site where an injection has been given. Additionally, the number of polio cases increased drastically after mass vaccinations against diphtheria and whooping cough in the 1940s, as documented in the Lancet and other publications.

Polio, like most diseases, may be conditional on various factors. It makes particular sense, however, to take poisoning by industrial and agricultural pollution into consideration, to explain why this nervous disease first appeared in the 19th century, in the course of industrialization. It spread like wildfire in the industrialized West in the first half of the 20th century, while in developing countries, in contrast, there was no outbreak.

In the 19th century, the disease was named poliomyelitis, referring to degeneration of spinal column nerves (myelitis is a disease of the spinal cord) typical of polio. Orthodox medical literature can offer no evidence that the poliovirus was anything other than benign until the first polio epidemic, which occurred in Sweden in 1887. This was 13 years after the invention of DDT in Germany (in 1874) and 14 years after the invention of the first mechanical crop sprayer, which was used to spray formulations of water, kerosene, soap and arsenic.
The epidemic also occurred immediately following an unprecedented flurry of pesticide innovations, says Jim West of New York, who has extensively investigated the subject of polio and pesticides. This is not to say that DDT was the actual cause of the first polio epidemic, as arsenic was then in widespread use and DDT is said to have been merely an academic exercise. However, DDT or any of several neurotoxic organochlorines already discovered could have caused the first polio epidemic if they had been used experimentally as a pesticide. DDTs absence from early literature is little assurance that it was not used.

Nearly ten years before, in 1878, Alfred Vulpian, a neurologist, had provided experimental evidence for the poisoning thesis when he discovered that dogs poisoned by lead suffered from the same symptoms as human polio victims. In 1883, the Russian Miezeyeski Popow showed that the same paralysis could be produced with arsenic. These studies should have aroused the scientific community, considering that the arsenic-based pesticide Paris green had been widely used in agriculture to fight pests like caterpillars since 1870.

But instead of prohibiting the insecticide Paris green, it was replaced by the even more toxic pesticide: lead arsenate, which likewise contained heavy metals, in the state of Massachusetts in 1892, according to a 2004 article in the British magazine The Ecologist. Indeed, a polio epidemic broke out in Massachusetts two years later. Dr. Charles Caverly, who was responsible for the tests, maintained that a toxin was more likely the culprit than a virus, stating emphatically that, we are very certainly not dealing with a contagious disease.

Within a short time, however, lead arsenate became the most important pesticide in the industrialized world’s fruit cultivation. It was not the only toxic substance used in agricultural industries. In 1907, for example, calcium arsenate was introduced in Massachusetts and was used in cotton fields and factories. Months later, 69 children who lived downstream from three cotton factories suddenly became sick and suffered from paralysis. Meanwhile, lead arsenate was also being sprayed on the fruit trees in their gardens. But microbe hunters ignored these legitimate cluster factors, and instead continued searching for a responsible virus.

A cornerstone for the polio-as-virus theory was laid down in 1908 by scientists Karl Landsteiner and Erwin Popper, both working in Austria. The World Health Organization calls their experiments one of the milestones in the obliteration of polio. That year, another polio epidemic occurred and once again there was clear evidence that toxic pesticides were at play. But, astoundingly, instead of following up this evidence, medical authorities viewed the pesticides as weapons in the battle against the arch enemy microbes. They even neglected to give the children suffering from lameness treatments to alleviate the pesticide poisoning and, thus establish whether their health could be improved this way. (In 1951, Irwin Eskwith did exactly that and succeeded in curing a child suffering cranial nerve damage-bulbar paralysis, a particularly severe form of polio-with dimercaprol, a detoxification substance that binds heavy metals like arsenic and lead).

Landsteiner and Popper instead chose to take a diseased piece of spinal marrow from a lame nine-year-old boy, chopped it up, dissolved it in water and injected one or two whole cups of it intraperitoneally (into the abdominal cavities) of two test monkeys: one died and the other
became permanently paralyzed. Their studies were plagued by a mind-boggling range of basic problems. First, the glop they poured into the animals was not even infectious, since the paralysis didn’t appear in the monkeys and guinea pigs given the alleged virus soup to drink, or in those that had it injected into their extremities. Shortly after, researchers Simon Flexner and Paul Lewis experimented with a comparable mixture, injecting this into monkey’s brains. Next, they brewed a new soup from the brains of these monkeys and put the mix into another monkeys head. This monkey did indeed become ill. In 1911, Flexner even boasted in a press release, that they had already found out how polio could be prevented, adding, of course, that they were close to developing a cure.

But this experiment shows no proof of a viral infection. The glop used cannot be termed an isolated virus, even with all the will in the world. Nobody could have seen any virus, as the electron microscope wasn’t invented until 1931. Also, Flexner and Lewis did not disclose the ingredients of their injection soup. By 1948, it was still unknown how the polio virus invades humans, as expert John Paul of Yale University stated at an international poliomyelitis congress in New York City.

Apart from that, it is very probable that the injection of foreign tissues in the monkey’s craniums triggered their polio-like symptoms. And when one considers the amount of injected material, it can hardly be surprising that the animals became ill. Controlled trials weren’t even carried out—that is, they neglected to inject a control group of monkeys with healthy spinal cord tissue. Neither were the effects of chemical toxins like heavy metals injected directly into the brain. All of these factors make the experiments virtually worthless.

Although many scientific factors spoke against the possibility that polio was an infectious viral disease, these studies would become the starting point of a decade long fight, which concentrated exclusively on an imaginary polio virus. Anything and everything, like brain parts, feces, and even flies were chased into the monkey’s brains in an attempt to establish a viral connection. Later these monkeys were even captured en masse in the Indian wilderness and transported overseas to the experimental laboratories—with the single aim of producing paralysis. And where virus hunters were working, vaccine manufacturers were not far away.

By the end of the 1930s, vaccine researchers had allegedly discovered a whole range of virus isolates. But these could not have been real isolates. And another problem cropped up along the way: the monkeys didn’t get sick when they were orally administered the glop. These researchers could only produce paralysis by injecting into the brain large amounts of substrates of unknown contents. In 1941, the polio virus hunters had to accept a bitter setback, when experts reported in the scientific journal Archives of Pediatrics that, Human poliomyelitis has not been shown conclusively to be a contagious disease. Neither has the experimental animal disease, produced by the so-called poliomyelitis virus, been shown to be communicable. In 1921, Rosenau stated that monkeys have so far never been known to contract the disease spontaneously even though they are kept in intimate association with infected monkeys. This means that if this was not an infectious disease, no virus could be responsible for it, so the search for a vaccine was a redundant venture.

But virus hunters didn’t even consider factors that lay outside of their virus obsession. So it
happened that, in the middle of the 20th century, researcher Jonas Salk believed he had conclusively found the polio virus. Even though he could not prove that what he called the polio virus actually triggered polio in humans, he still somehow believed he could produce a vaccine from it.

Salk alone is said to have sacrificed 17,000 test monkeys (termed the heroes by one of Stalks co-workers) on the altar of vaccine research during the most heated phase of his research; in total, the number of slaughtered monkeys reached into the hundreds-of-thousands. But critics objected that what Salk termed the polio virus was simply an artificial product of the laboratory.

Consequently, to this day, it is a huge challenge to find what is termed the polio virus where the patient’s nerve cells are damaged, that is to say, in spinal cord tissue. In 1954, Bernice Eddy, who was then responsible from the US governments vaccine safety tests, also reported that the Salk vaccine had caused severe paralysis in test monkeys. Eddy was not sure what had triggered the paralysis symptoms: a virus, some other cellular debris, a chemical toxin? But it contained something that could kill. She photographed the monkeys and submitted them to her boss-but he turned her down and criticized her for creating panic. Instead, of course, he should have taken her misgivings into account and started extensive inquiries. But Eddy was stopped by the microbe establishment and even had to give up her polio research shortly before her warnings had proven themselves justified.

On 12 April 1955, Salk’s vaccine was celebrated nationwide as a substance that completely protected against polio outbreaks. US President Dwight Eisenhower awarded Salk a Congressional Gold Medal. American and Canadian television joined in the celebration. And on 16 April, the Manchester Guardian joined the party, stating that nothing short of the overthrow of the Communist regime in the Soviet Union could bring such rejoicing to the hearths and homes in America as the historic announcement last Tuesday that the 166-year war against paralytic poliomyelitis is almost certainly at an end.

But the triumph was short-lived. Medical historian Beddow Bayly wrote that Only thirteen days after the vaccine had been acclaimed by the whole of the American Press and Radio as one of the greatest medical discoveries of the century, and two days after the English Minister of Health had announced he would go right ahead with the manufacture of the vaccine, came the first news of disaster. Children inoculated with one brand of vaccine had developed poliomyelitis. In the following days more and more cases were reported, some of them after inoculation with other brands of the vaccine. According to Bayly, Then came another and wholly unlooked-for complication. The Denver Medical Officer, Dr. Florio announced the development of what he called satellite polio, that is, cases of the disease in the parents or other close contacts of children who had been inoculated and after a few days illness in hospital, had returned home [and] communicated the disease to others, although not suffering from it themselves.

Within only two weeks, the number of polio cases among vaccinated children had climbed to nearly 200. On 6 May 1955, the News Chronicle quoted the US governments highest authority on viruses, Carl Eklund, who said that in the country, only vaccinated children, had been afflicted by polio. And only, in fact, in areas where no polio cases had been reported for a good three-quarters of a year. At the same time, in nine out of ten cases, the paralysis appeared in the
injected arm. This triggered panic in the White House. On 8 May, the American government completely halted production of the vaccine. A short time later, a further 2,000 polio cases were reported in Boston, where thousands had been vaccinated. In inoculated New York, the number of cases doubled, in Rhode Island and Wisconsin, they jumped by 500%. And here as well, the lameness appeared in the inoculated arm in many children.

Apart from that, an objective look at statistics would have shown that there was no reason to celebrate Salk’s vaccine as the great conqueror of an alleged polio virus. According to international mortality statistics, from 1923 to 1953, before the Salk killed-virus vaccine was introduced, the polio death rate in the United States and England had already declined on its own by 47% and 55% respectively, writes scientific journalist Neil Miller. In the Philippines, only a few years before the US catastrophe, the first polio epidemic in the tropics occurred spontaneously, in fact, with the introduction of the insecticide DDT there.

Around the end of World War II, US troops in the Philippines had sprayed masses of DDT daily to wipe out flies. Just two years later, the well-known Journal of the American Medical Association reported that lameness among soldiers stationed in the Philippines could not be differentiated from polio, and it had advanced to become the second most common cause of death. Only combat exercises were said to have claimed more victims. Meantime, populations in neighboring areas, where the poison had not been sprayed, experienced no problems with paralysis. This is further evidence that DDT poisoning can cause the same clinical symptoms as polio (which is claimed to be conditional upon a virus). Young people in industrialized countries are hardly acquainted with DDT anymore. It stands for dichlorodiphenyltrichloroethane, and is a highly toxic substance first synthesized at the end of the 19th century, in 1874, by Austrian chemist Othmar Zeidler. Paul Hermann Muller of Switzerland discovered its insect killing property in 1939, for which he received the Nobel Prize for Medicine in 1948. This resulted in its widespread use for pest control, even though there was already strong evidence that it was a severe neurotoxin, dangerous for all forms of life and associated with the development of herpes zoster (shingles), produces paralysis, has carcinogenic potential and can be fatal.

DDT is also problematic because it biodegrades very slowly in nature with a half-life of 10-20 years. Additionally, through the food chain, it can become concentrated in the fatty tissue of humans and animals. But this toxic substance wasn’t outlawed until 1972 in the USA and even later in most other countries in the prosperous northern hemisphere. Today, its use is prohibited in a large part of the world and it one of the dirty dozen organic toxins banned worldwide at the Stockholm Convention on 22 May 2001.

Industrial production of DDT started at the beginning of the 1940s. It was first used to fight malaria, and later became a sort of all-purpose remedy against all sorts of insects. There was also military use of DDT. US army recruits were powdered with it to protect them from lice, and they additionally received DDT sprayed shirts. m When the Second World War was over, DDT was sold on stock markets round the globe, even though strong warnings about its toxicity had been issued. In the mid-40s, for example, the National Institutes of Health demonstrated that DDT evidently damaged the same part of the spinal cord as polio, writes research scientist Jim West of New York.
The classic Harrison’s Principle of Internal Medicine states, Lameness resulting from heavy metal poisoning is clinically sometimes difficult to differentiate from polio. Endocrinologist Morton Biskind came to the same conclusion in his research papers describing the physiological evidence of DDT poisoning that resembles polio physiology: Particularly relevant to recent aspects of this problem are neglected studies by Lillie and his collaborators of the National Institutes of Health, published in 1944 and 1947 respectively, which showed that DDT may produce degeneration of the anterior horn cells of the spinal cord in animals. These changes do not occur regularly in exposed animals any more than they do in human beings, but they do appear often enough to be significant.

Biskind concludes: When in 1945 DDT was released for use by the general public in the United States and other countries, an impressive background of toxicological investigations had already shown beyond doubt that this compound was dangerous for all animal life from insects to mammals.

Despite the fact that DDT is highly toxic for all types of animals, the myth has spread that it is harmless, even in very high doses. It was used in many households with a carefree lack of restraint, contaminating people’s skin, their beds, kitchens and gardens. In Siskind’s opinion, the spread of polio after the Second World War was caused by the most intensive campaign of mass poisoning in known human history.

Along with DDT, the much more poisonous DDE was also used in the USA. Both toxins are known to break through the hematoencephalic barrier, which protects the brain from poisons or harmful substances. Nonetheless, housewives were urged to spray both DDT and DDE to prevent the appearance of polio. Even the wallpaper in children’s rooms was soaked in DDT before it was glued on the wall.

What from today’s perspective seems like total blindness was at that time an everyday practice, not only in the United States. After 1945, DDT powder was used in Germany to fight a type of louse said to carry typhus. And in agriculture, including fruit and vegetable cultivation, DDT was likewise lavishly dispersed for so-called plant protection. Through this, DDT gradually replaced its predecessor, lead arsenate, a pesticide containing heavy metals.

A look at statistics shows that the polio epidemic in the USA reached its peak in 1952, and from then on rapidly declined. We have seen that this cannot be explained by the Salk-inoculation, since this was first introduced in 1955. There is a most striking parallel between polio development and the utilization of the severe neurotoxin DDT and other highly toxic pesticides like BHC (lindane), which was also hard to degrade and actually much more poisonous than DDT. While use of DDT was eventually drastically reduced because of its extreme harmfulness, the use of BHC was curbed because it produced a bad taste in foods.

It is worth noting that DDT production rose dramatically in the United States after 1954, Jim West remarks, which is primarily connected to the fact that DDT was increasingly exported to the Third World, to be used primarily in programs to fight malaria or in agriculture. As West points out, the following factors contributed to its changed use patterns in the US:
1. An altered legislation led to the use of warning labels, which in turn raised public awareness of DDT's poisonous nature. Eventually, the use of DDT on dairy farms was prohibited. Earlier, Oswald Zimmerman and his fellow research scientists had even advised the daily spraying of a 5% DDT solution directly on cattle and pigs, their feed, drinking water, and resting places. In 1950, it was officially recommended to US farmers that they no longer wash cattle with DDT, but at first this advice was largely ignored. In the same year, cow’s milk contained up to twice as much DDT as is necessary to trigger serious illnesses (diseases) in humans.
3. In advertisements and press releases, DDT was no longer celebrated as being good for you, harmless, and a miracle substance.
4. From 1954, concentrated DDT was only used on crops that did not serve food production (for example, cotton).
5. DDT was used with more caution, something that caused decreased human intake of the poison through foodstuffs.
6. The use of DDT was extended to nationally sponsored forestry programs, so, for instance, entire forests were sprayed with it by airplane.
7. DDT was gradually replaced by allegedly safe pesticides in the form of organophosphates like malathion, but their uncertain toxicological effects and the new pesticide laws merely changed the type of neurological damage from acute paralysis to less-paralytic forms, such as chronic, slow-developing diseases which were difficult to define. This made it particularly difficult to prove in legal disputes or studies that these pesticides contributed to or directly caused the illnesses in question: BSE as an Effect of Chemical Poisoning for more on the organophosphate phosmet).

Finally in 1962, US biologist Rachel Carson published her book, Silent Spring, in which she gives a vivid account of the fatal repercussions of extensive spraying of plant toxins on insects and particularly on birds, and predicts the consequence of a silent spring (without any songbirds). Through this, the public was made aware of the dangers of DDT. But public reaction was slow, because 800 chemical companies reacted hysterically to Carson’s book, prophesying hunger and destruction if farmers were no longer permitted to use any pesticides. The goal was very obviously to create panic and drive farmers into the arms of the chemical industry, as Pete Daniel, expert on the history of pesticides, writes in his 2005 book, Toxic Drift. In 1964, a North Carolina turkey breeder named Kenneth Lynch wrote to the Ministry of Health, stating that, since 1957, his home town of Summerville had been enveloped in a mist of DDT or malathion (an insecticide which can have wide-ranging neurotoxic and fatal effects) 150 every summer, in order to kill mosquitoes.

And over the past years, his turkeys had more or less abruptly developed advanced paralyses and, even though they had originally been in good health, died within two or three days. At the same time, the fertility of the eggs had declined from 75% to 10%. The evidence clearly indicated that the fog of insecticide is to blame, writes Lynch. With the help of a chemistry professor, he turned to the Public Health Service (PHS) and suggested carrying out corresponding studies. The national authorities, however, showed no interest whatsoever. It seems to me [that the ministry’s behavior] can hardly be interpreted as anything other than a case
of bureaucracy being blinded by its own past mistakes, opined Clarence Cottam, a biologist honored by the National Wildlife Federation as a protector of nature.

In their refusal, political decision-makers and the chemical industry’s lobbyists referred primarily to the prisoner studies of PHS scientist Wayland Hayes. In these experiments on prisoners, Hayes had aimed to show that it was completely harmless to ingest 35 milligrams of DDT per day. But critics like Cottam objected that every test subject could release him/herself from the experiments at any time. And indeed, there were a fair number who withdrew when they became a bit ill.

Since a number of prisoner test patients dropped out of the study, data on adverse effects were largely eliminated, so the study’s results were worthless. Cottam points out that Hayes had most likely engaged in researcher bias to substantiate his initial views on pesticides: Perhaps he is like many human beings who when subjected to criticism become more and more dogmatic in maintaining their initial stand.

Pesticide historian Pete Daniel goes a step further in saying that [the officials in charge] knew better, but the bureaucratic imperative to protect pesticides led the division into territory alien to honesty. It would be years before the US government held a hearing on DDT and even longer until they finally prohibited it in 1972. Unfortunately, the government discussions were not widely reported, so the general public remained unaware of the connection between polio (in humans!) and pesticides, and other non-viral factors. To achieve this at the beginning of the 1950s ten years before Carson’s Silent Spring, someone would have had to have written a bestseller which described the repercussions of DDT (and other toxins) in humans.

Unfortunately, this was not the case; and it was not until this book, Virus Mania, was published. Carson’s book was good, but it was restricted to the damage to animals, whereas one looks in vain for descriptions of statistical trends or analyses in the work, says Jim West. Even the research scientists Biskind and Scobey, who had clearly described the damage that DDT causes in humans, were practically unmentioned by Carson. Now who knows what kind of editorial censoring process her book had to go through before its publication.

West points out that this type of censorship became the norm in future virus research: One needs only consider that her work had been financed by the Rockefeller Foundation. This makes one sit up and take notice, for the Rockefeller Foundation has supported the significant orthodox epidemic programs, including the HN = AIDS research and numerous vaccination programs. And the great Grandfather Rockefeller had made his money by selling snake venom and pure mineral oil as a universal cure. Carson’s book prompted public outcry, which contributed to DDT’s ultimate prohibition. But this was a deceptive victory, which only helped to secure the public belief that democratic regulative mechanisms still functioned effectively. In actual fact, the chemical industry—because the public thought the poisonous demon had then been defeated—was able to establish its likewise highly toxic organophosphate on the market without a problem. And, fatally, nobody discussed its important central topic: that poisons like DDT could cause severe damage like polio.

The virus hunters still had many weapons to pull from their box of tricks. Such as the concept of
the slow virus: a virus capable of sleeping in a cell for years before striking with its pathogenic or fatal effects. The claim that a disease takes a very long time (decades) to break out gained popularity in the 1960s, when virus hunters convinced the medical establishment that the virus concept could even be imposed on cancer that is, a disease that generally appears after years or decades.

But despite a most arduous search, researchers were simply unable to find any active viruses in tumors. The disappointment and frustration was correspondingly great. But a new theory was soon developed: that a virus could provoke an infection, and then lie dormant in a cell for as long as it wanted—and finally, at some point, even trigger cancer, and even when the virus is no longer present. Just as with polio earlier, the nucleic acids of a so-called slow virus have never been isolated and the particles have never been imaged with an electron microscope, but the virus hunters embraced this suspect theory and adapted it to a number of modern ailments.

· Scientist Carleton Gajdusek prodded the slow virus concept along to serve not only an explanatory model for HN I AIDS. In the 1970s in Papua New Guinea, Gajdusek researched a sponge-like alteration in brain tissue associated with dementia, which was predominantly spread among the female population there. The disease, called kuru, was only observed in two clans; they often intermarried, and, according to Gajdusek, maintained a cult of the dead ritual that involved eating the brains of their deceased (something which was later revealed as a myth). These transmissible spongiform encephalopathies (softening of the brain), as they are called, appear sporadically and end, mostly fatally, within five years. They are generally extremely rare (approximately one case per million people), but are represented within some families with a frequency of 1 in 50, which could point to a genetic cause. Despite this Gajdusek received the Nobel Prize in 1976 for his slow virus concept. With this endorsement his idea that this sponge like alteration in brain tissue was produced and transmitted by a pathogen achieved widespread acceptance as fact.

A close look at Gajdusek’s trials on apes, with which he aimed to show transmissibility, should have shocked the scientific community into disbelief. But instead, they recognized these papers as proof of transmissibility and ignored the fact that neither feeding the ape’s brain mush, nor injecting them with it had any effect on the chimpanzees. So, Gajdusek conducted a bizarre experiment, in order to finally induce neural symptoms in the test animals.

He ground up the brain of a kuru patient into a mush full of proteins, along with a number of other substances, and poured this into the living apes by drilling holes into their skulls. This so-called diseases alleged transmissibility was founded only upon these experiments. How could it possibly derive proof of Gajdusek’s cannibalistic hypothesis? Particularly since the hypothesis indicates that the disease could appear in humans through ingestion of infected brains, and not through direct surgical insertion into the brain.

To compound matters, Gajdusek was the only living witness of cannibalism on Papua New Guinea. He reported on these cannibalistic rites in his 1976 Nobel Prizewinning lecture, even documenting them with photographs. But in the mid-1980s, it was discovered that Gajdusek’s photos, with which he aimed to document the cannibalism, actually showed pig flesh, not human
flesh. An anthropological team looked into this claim and they did find stories of cannibalism, but no authentic cases.

Gajdusek later had to admit that neither he himself, nor others he met had seen the cannibalistic rites. Roland Scholz, Munich-based professor of biochemistry and cellular biology in Munich, responded to this revelation by saying that, the scientific world seems to have been taken in by a myth.

Modern viral research is like Bigfoot hunting. Trackers of this legendary ape-like beast (also called Sasquatch and the Abominable Snowman) trot out the occasional questionable blurry photograph and footprint marks to claim proof of Bigfoot’s existence. Based on this suspect data, they say the beast is up to ten feet tall and 440 pounds with 17-inch footprints that have even been made into plaster casts to prove its existence. Virus hunters also collect dubious data, claiming to have images of the virus, even though electron micrographs of viruses accompanied by an analysis of their complete genetic material and virus shell are the only method of proving a virus’s existence.

Bigfoot hunt, like viruses, are splendid moneymakers. Along a strip of California’s Highway 101, numerous shops hawk Bigfoot-souvenirs and they are popular with tourists even though it is generally accepted that Bigfoot is an invention. Of course, Bigfoot is nowhere near as lucrative as the international virus industry’s multi-billion dollar business.

We must stress here that electron microscopy is fundamental to virus identification. For a long time, establishing unequivocal proof of a virus meant seeing believes, as is the case with bacteria and fungi. The one difference is that bacteria and fungi can be seen with a light microscope, whereas viruses are so tiny that only an electron microscope (first patented in 1931) enables detailed imaging to make them visible. But, first you have to identify exactly what you’re looking at, so these particles (possible viruses) must exist in a pure or purified form, in order to be able to differentiate virus particles from virus-like ones. At the beginning of the 1950s, virologists agreed that this was necessary, since, under certain conditions, even healthy cells produce a whole range of particles that could look like so-called tumor viruses (oncoviruses).

The importance of this process was confirmed at an international meeting of the Pasteur Institute in 1972, and endured in the early 1980s, according to Val Turner, a physician and member of the Perth Group, an Australian research team. Viruses are not naked bits of RNA (or DNA).

They are particles with particular sizes and shapes and other identifying features, which are obliged to replicate at the behest of living cells. They won’t multiply in dead meat like bacteria. So there you have it. This predicates experiments to prove particles are a virus and that hasn’t changed in a thousand years and certainly not since the 90s.

Turner uses easy-to-grasp language to describe the science: Think of it like a paternity suit in which DNA evidence will be used and the accused is HIV and the child is a human. The crux of the case is proof that the DNA you found in the human is the same DNA you found in the accused. For the latter, you have to have rock solid proof the DNA carne from the accused. Given that in cell cultures all sorts of particles appear, only some of which are viruses, you have to prove that (a) a particular particle is a virus; and (b) your DNA comes from that particle. How
can you prove (a) without using electron microscopy (for many reasons) and without purification? You tell me.

Frankly we from the Perth Group do not understand this obsession with old data or science moves on. Has Archimedes principle moved on? Do solid objects no longer displace their own volume of liquids? If everything has to be up to date then in ten years nothing that is up to date now will be up to date then. Which means as long as time keeps going nothing will be right. This goes for orthodox theories as well!

By soundly characterizing virus structure (virus purification), it is theoretically possible to irrefutably differentiate viruses themselves from virus-like particles. If this has taken place, the next step would be to get an electron micrograph of the purified virus (of course, proof that a virus exists does not automatically mean that this virus is also infectious, as had already been established in 1960, at a conference sponsored by the New York Academy of Sciences). But this procedure is rarely carried out in modern viral research. Viruses that purportedly threaten to wipe out humanity (H5N1, SARS virus, etc.) have evidently never been seen by anyone. Around 1960, before contemporary molecular biology arose, electron microscopy was held to be the best way of identifying viruses in cell cultures, writes pathology professor Etienne de Harven, a pioneer in electron microscopy and virology. De Harvens research career includes 25 years at the Sloan-Kettering Institute in New York, a private cancer research center founded in 1945, which quickly advanced to become the largest of its kind in the USA. For this reason, laboratories all over the world directed their efforts at this time towards observing particles in cancer cells with ever-improved methods of electron microscopy. In 1962, the central role of electron microscopy was also recognized at the well-known Cold Spring Harbor Conference. Andre Lwoff, who would receive the Nobel Prize for medicine three years later, was among those who designated electron microscopy as likely the most efficient method of proving viruses existence; he suggested investigating viruses with this procedure and dividing them into classes.

A focus of medical science then (as now) was cancer. And because cancer researchers had the fixed idea that viruses were definitely cancer triggers, they spent a lot of time proving the presence of viruses in human cancer cells, with the help of electron microscopy. But, these efforts were unsuccessful. One only found virus-like particles from time to time-while viruses of a certain types could never convincingly be seen, reports de Harven.

Virus hunters were, once again, crushed by this scientific news. But the scientific world tends not to publicize negative results whenever possible-in scientific language, this is called, publication bias. Yet, whether the research claims promoted as evidence involve new patented drugs said to be superior to existing (cheaper) ones, or genetic markers of disease (interpreted as risk factors) , or statistical relationships, discerning whether the claims are spurious or confirmed by clinical trials can only be ascertained by making the full body of controlled studies publicly available.

In medicine, failure to do so casts doubt on the safety and efficacy of treatments as well as undermining the integrity of the scientific literature. Scientific journals are supposed to protect the integrity of science—but they don’t. As is the case with most deficient practices in medical research and practice, there is an unacknowledged financial motive. And why are scientists coy about publishing negative data? In some cases, says Scott Kern of Johns Hopkins University and
editor of the recently founded online Journal of Negative Observations in Genetic Oncology, withholding them keeps rivals doing studies that rest on an erroneous premise, thus clearing the field for the team that knows that, say, gene A doesn’t really cause disease B. Which goes to show that in scientific journals, no less than in supermarket tabloids, you can’t believe everything you read—or shouldn’t.

As long ago as the 1960s the established science community was coy about publishing negative data, but the cancer virus hunters failures were so universal that it was simply inevitable that one article or another should leak out into medical publications. In 1959, the researcher Hagenaus reported in the journal Etude du Cancer about the difficulties identifying any typical virus particles in a wide range of breast cancer samples. And in 1964, the scientists Bernhard and Leplus were unsuccessful, even with electron microscopy’s assistance, in finding virus particles presumed to play a role in the development of Hodgkin’s lymphoma (lymphatic cancer), lymphoid leukemia or metastases (tumors in various parts of the body).

But these scientific studies didn’t stop the virus hunters for a second. Instead of disengaging themselves from their virus tunnel vision, they grumbled about the methodology of virus determination: for example, over what are known as thin slices or thin-sections (tissue samples which are extremely precisely dissected and trimmed to size so they can be observed under the electron microscope). Thin sections had proved effective countless times, and had also worked perfectly with mice. But, the virus hunters needed a scapegoat and, instead of questioning the cancer-producing virus model, they started griping about the thin-sections. The production of the thin-sections was also thought to be too laborious and time-consuming. And who had the time for that once pharmaceutical companies began offering fast cash for quick fixes?

So, scientists turned to the much simpler and faster dye method, in which certain particles of the sample (for instance, DNA and RNA) were marked in color and then electron micrographed. But from a purely scientific perspective, the results of dye method are a disaster. Through the air-drying process that was necessary for the staining, the particles became totally deformed, so that they appeared as particles with long tails. They were full-blown artificial products of the laboratory, and they still looked exactly like so many other non-viral cellular components. This, logically, made it impossible to determine if a virus or a non-viral particle had been found.

A few scientists did in fact acknowledge that the dye method was dubious. But, instead of admitting defeat and returning to the thin-sections method, they began bashing electron microscopy technology! Other researchers were in turn so anxiously preoccupied with finally finding cancer viruses that they casually overlooked the worthlessness of dye method results, and theorized that the tailed particles were a certain type of virus. As absurd as this may sound to logical thinkers, virus hunters were even remunerated with plenty of research money for this action.

As a result, even cow’s milk and mother’s milk were tested for the presence of tailed particles in the mad rush to prove that viruses could produce cancer. One well-known molecular biologist Sol Spiegelman even warned against breastfeeding in October 1971, and his message made for numerous lurid media headlines.
These so-called scientists brushed aside the fact that, to date, not a single retrovirus has been able to be isolated from breast cancer tissue (and probably not from human tumor tissue or blood plasma in general). Shortly thereafter, Spiegelman was quoted in Science saying, one can’t kick off fear mongering on this scale if one doesn’t exactly know if a virus particle is the cause.

But mainstream viral research drifted purposefully further away from the well-established viral proof model. They latched on to Howard Temins and David Baltimore’s description of activity of the enzyme reverse transcriptase in connection with cancer viruses in 1970. Their research seemed so significant to the medical establishment that the two were awarded the Nobel Prize in 1975.

What was so significant about this enzyme, a substance that, as a sort of catalyst, makes it possible for biochemical reactions to occur? To understand this, we must remember that, in the 1960s, scientists thought they had established that a few viruses did not possess any DNA (complete genetic information), but rather only RNA genes. This baffled the researchers since they believed viruses without any DNA (only with RNA) were not able to multiply. Until Temin and Baltimore delivered an explanation with the enzyme called reverse transcriptase. It, they said, can transform the RNA in RNA viruses (later called retroviruses because of this) into DNA, by which viruses are then able to multiply (if RNA exists alone, the conditions for replication are not met).

But there was so much enthusiasm about the discovery of reverse transcriptase that virus hunters rashly assumed that reverse transcriptase was something very typical of retroviruses. They proclaimed something like this: if we observe reverse transcriptase activities in our test tubes (in vitro), then we can be sure that a retrovirus is present as well (even if the virus existence has never been proven or reverse transcriptase’s role hasn’t been established, for instance, in the context of HIV). Yet, it was presumed that the (indirectly detected) presence of reverse transcriptase was sufficient enough to prove the existence of a retrovirus, and even a viral infection of the tested cells in vitro.

This dogma would now become fixed in the minds of mainstream researchers and it opened the floodgates to allow indirect virus detection methods (known as surrogate markers) to take the place of direct detection procedures (virus purification and characterization as well as electron micrograph).

So, in 1983, in a paper printed in Science, researcher Luc Montagnier of the Institute Pasteur in Paris, later celebrated as the discoverer of HN, asserted that his research team had found a new retrovirus (which would later be named HN).

This was claimed only after reverse transcriptase activity had been observed in the cell culture. But, once again, there was no scientific proof for this conclusion. Eleven years before, in 1972, Temin and Baltimore had stated, reverse transcriptase is a property that is innate to all cells and is not restricted to retroviruses. And even Françoise Barre-Sinoussi and Jean Claude Chermann, the most important co-authors of Montagniers 1983 Science paper, concluded in 1973 that reverse transcriptase is not specific to retroviruses, but rather exists in all cells.
In other words, if the enzyme (the surrogate marker) reverse transcriptase is found in the laboratory cultures, one cannot conclude, as Luc Montagnier did, that a retroviruses, let alone a particular retrovirus has been found.

Reverse transcriptase is no longer the most significant surrogate marker, by a long shot. Now the virus hunters are fixated on antibody tests, PCR viral load tests, and helper cell counts. But these tests raise new questions, given their striking weaknesses. This prompted renowned virologists of the old guard to direct an appeal to the young high-technology-focused generation of researchers, which was published in Science in 2001: Modern methods like PCR, with which small genetic sequences are multiplied and detected, are marvelous [but they] tell little or nothing about how a virus multiplies, which animals carry it, how it makes people sick. It is like trying to say whether somebody has bad breath by looking at his fingerprint.

No less remarkable, in this context, is an early 2006 article in the German Medical Journal about a study by researchers who thought that, with the assistance of PCR, they had discovered new exotic bacteria. The article points out that, only genetic traces of the pathogen are detected [with the PCR]. From this, it cannot automatically be concluded that complete bacteria exist as well.

Among the overall virus mania, such critical thoughts founder quickly. In the 70s, elite researchers were simply too busy channeling generous government aid into researching the possible connection between viruses and cancer. On 23 December 1971, US President Richard Nixon declared the War on Cancer at the behest of the medical establishment, and, with this metaphor, carried the militant tradition of the monocausal medical doctrine to the extreme, attached to the conception of viruses as the enemy. We had now become accustomed to talking about the weapons, the strategies, and the arsenals of cell-killing preparations and weren’t even taken aback when powerful people like Nixon called the new cancer war a Christmas present for the people.

To date, many hundred millions of dollars of research funds have been poured into this war (a good part of it paid by taxes)—and the results are staggering. Back in 1971, a cure for cancer and a preventive vaccine were promised by 1976—but both of these are still nowhere in sight.

Incidentally, in the tradition of celebratory medicine, along with a trust that the public conscience and the media have short-term memory, the medical establishment rarely feels a need to keep its promises. I am convinced that in the next decade or maybe later, we will have a medication that is just as effective against cancer as penicillin against bacterial infections, boasted Cornelius Dusty Rhoads as early as 1953. He had been leader of the US Army’s Department for Chemical Warfare (medical division of the US Chemical Warfare branch) during the Second World War, and was director of the Sloan-Kettering Institute for Cancer Research, founded in 1945.

Death rates have meantime increased exponentially alongside skyrocketing research expenditures. Today in Germany, 220,000 people die annually from cancer; in the USA, it is almost 600,000. Even taking the aging of these populations into consideration, these numbers are staggering. For this reason, experts like George Miklos, one of the most renowned geneticists
worldwide, criticized mainstream cancer research in Nature Biotechnology as fundamentally flawed and equated it with voodoo science.

By the late 1970s, medical experts lobbed damning critiques against mainstream cancer research. Medical scientists had credited the retroviruses with every nasty thing—above all the triggering of cancer—and have to accept constant mockery and countless defeats, Der Spiegel pointed out in 1986.

And the concept that viruses are the great trigger factors failed with other diseases, besides cancer. One notorious example is the swine flu disaster of 1976. During a march, David Lewis, a young American recruit, collapsed. Epidemic experts swooped in with their magic wand of clustering in their hands and claimed that they had isolated a swine flu virus from his lung. At the behest of the medical establishment, and particularly the US Centers for Disease Control (CDC), US President Gerald Ford appeared on TV and urged all Americans to get vaccinated against an imminent deadly swine flu epidemic. Just like today’s avian flu fear mongers, Ford used the great Spanish flu pandemic of 1918 to scare the public into action.

Approximately 50 million US citizens rushed to local health centers for injections of a substance hastily thrown on the market. It produced strong side effects in 20% to 40% of recipients, including paralysis and even death. Consequent damage claims climbed to $2.7 billion. In the end, CDC director David Spencer, who had even set up a swine flu war room to bolster public and media support, lost his job. The ultimate bitter irony was that there were no, or only very isolated reports of swine flu.

Consequently, at the end of the 1970s the US National Institutes of Health (NIH) came into unsettled political waters—just like the CDC, which was extensively restructured at the beginning of the 1980s. As a result, at the CDC and NIH, the most powerful organizations related to health politics and biomedical science, the great contemplation began. To redeem themselves, a new war would, of course, be the best thing.

Despite perpetual setbacks, an infectious disease remained the most effective way to catch public attention and open government pockets. In fact, Red Cross officer Paul Cumming told the San Francisco Chronicle in 1994 that the CDC increasingly needed a major epidemic at the beginning of the 80s to justify its existence. And the HIV 1 AIDS theory was a salvation for American epidemic authorities.

All the old virus hunters from the National Cancer Institute put new signs on their doors and became AIDS researchers. [US President Ronald] Reagan sent up about a billion dollars just for starters, according to Kary Mullis, Nobel laureate for Chemistry. And suddenly everybody who could claim to be any kind of medical scientist and who hadn’t had anything much to do lately was fully employed. They still are.

Among those who jumped over from cancer research to AIDS research, the best known is Robert Gallo. Along with Montagnier, Gallo is considered to be the discoverer of the AIDS virus, enjoys worldwide fame, and has become a millionaire. In his previous life as a cancer researcher, on the other hand, he had almost lost his reputation, after his viral hypotheses on diseases like leukemia
imploded. HIV didn’t suddenly pop out of the rain forest or Haiti, writes Mullis. It just popped into Bob Gallos hands at a time when he needed a new career.

Chapter 3
Whoever experienced the 1980s will still clearly remember: The AIDS panic picked up so quickly that there was no time for a survey of the facts. The media stimulated fear of viruses had left behind such traces in society, as the German weekly newspaper Die Zeit wrote in 1990, that social psychologists even trace the imminent comeback of men’s white underwear [as a symbol of HN-and with that sterility right into the most intimate zones] back to the AIDS effect.

In 1984, Der Spiegel announced that, by the middle of the 1990s, the last German would become ill from AIDS, dying from it two years later (in other words: by the mid-1990s, AIDS would wipe out the entire German population). The magazine Bild der Wissenschaft made the same deadly predictions the following year (1985). In comparison, a 1986 forecast in US magazine Newsweek sounded moderate: by 1991, five to 10 million Americans would be infected by HN.

In reality, yearly, no more than a few hundred Germans die from AIDS. Moreover, these people actually die from traditional diseases (like lymphatic cancer or tuberculosis), which are then redefined as AIDS (see below: What is AIDS?). And as for Newsweek’s visions of horror: its prognosis was around ten times the 750,000 HN cases identified by US authorities.

750,000 is actually a cumulative number, since AIDS cases aren’t tracked yearly, meaning that number represents the total numbers since official AIDS records were started in the early 1980s. Obviously, with such a method of measurement, the figures appear many times scarier than they actually are. Additionally, logic dictates that such numbers can only increase, even if the number of new cases had gone down in a given year. Incidentally, only AIDS cases are counted cumulatively. Have you ever heard the evening news give the number of traffic accident deaths since the beginning of statistical records (and not just the deaths for a given year)? Certainly not.

Strangely, the Robert Koch Institute even admits that they proceeded this way: To catch the public’s attention and encourage a political readiness to act, large numbers were naturally more suitable. A trick in the presentation of AIDS cases, applied internationally at the time, served to do this: in the first years, in contrast to other diseases where the number of new cases each year is given (incidence), AIDS cases were accumulated from year to year (cumulative incidence). Anyone who impartially dives into the topic of HIV/AIDS, perpetually trips over such oddities, inconsistencies and contradictions-and searches in vain for scientific proof of the theory’s basic hypotheses: that a virus called HN causes AIDS. At the same time, we are dealing with a very complex topic, so to make the controversies around the study of the cause of AIDS understandable, we will begin with a section which compactly explains why doubts that HN exists and causes AIDS are justified and why it makes sense to name factors like drug consumption or malnutrition as causes of AIDS, or better: of the many diseases grouped together under the term AIDS.

Even the definition of AIDS (Acquired Immune Deficiency Syndrome) is anything but coherent. In contrast to other diseases, there is no universal definition of AIDS that could be used as a basis for sound statistics. For developing nations, for instance, the World Health Organization
WHO introduced the Bangui Definition in 1986, with which many patients have been diagnosed with AIDS. According to this definition, anyone suffering from a few common and non-specific symptoms, like weight loss plus diarrhea and itching, is declared an AIDS patient (without blood tests, and thereby without HN antibody tests). In poor countries like in Africa, where today a third of the population is undernourished, these symptoms are a well known mass phenomena.

In comparison, in wealthy countries like the USA and Germany, people are declared to be AIDS patients if they have tested positive in an antibody test, and simultaneously suffer from at least one of 26-likewise well known-diseases, including the vascular tumor called Kaposis sarcoma (KS), Hodgkin’s disease, herpes zoster (shingles) or tuberculosis. If a patient has a negative antibody test and KS, they have KS. If, on the other hand, a patient tests positive and has KS, they are an AIDS patient. But this type of definition is misleading— it is circular, since it is based on dubious, doubtful, unproven assumptions that HN exists; that HN can cause AIDS (or a disease like KS or herpes zoster); that a positive antibody test proves the existence of HN, and so on.

This HN is said to belong to a certain class of viruses called retroviruses. In order to prove, then, that HN is a specific retrovirus, it would first be necessary to have HN as a pure virus available, so that it can be imaged in a purified form with an electron microscope. But all electron micrographs of so-called HN taken from the mid-80s on, come, not from a patient’s blood, but from souped-up cell cultures. In some cases the cells have been cooked up for a week in a lab Petri dish. So-called AIDS experts didn’t even try to make scientific sense of their co-culturing techniques until 1997, when Hans Gelderblom, of the Robert Koch Institute in Berlin, took a stab at it.

But Gelderbloms article, published in the magazine Virology, leaves out the purification and characterization of a virus (merely the protein was found), which does not prove that the particles are HN. The second image of patient’s blood came from the American National Cancer Institute. But the particles made visible (proteins, RNA particles) did not have morphology typical of retroviruses (let alone of a specific retrovirus). Additionally, proteins like and, which, according to the opinions of mainstream AIDS researchers, are supposed to be specific to HN, and are also used as HN markers (surrogate markers), were found in a number of so-called uninfected human tissue samples.

Even Luc Montagnier, called the discoverer of HN, admitted in an interview with the journal Continuum in 1997 that even after Roman effort, with electron micrographs of the cell culture, with which HN was said to have been detected, no particles were visible with morphology typical of retroviruses.

If even retrovirus-like particles cannot be recognized in these electron micrographs (let alone particles that match a retrovirus or a very particular retrovirus), this must logically mean that HN—allegedly, a very specific retrovirus—cannot be detected. Indeed, HN has never been detected in a purified form, according to many renowned experts, including Etienne de Harven, the previously mentioned pioneer in electron microscopy and virology, and AIDS researchers Eleni Papadopulos and Val Turner of the Australian Perth Group.
Nonetheless, in 2006, it was proudly reported once again that the structure of the world’s most deadly virus had been decoded and that HN had been photographed in a 3-D quality never achieved before. But a close inspection of the British-German research teams paper (published in the journal Structure), shows that it doesn’t live up to its promises:

- Firstly, it must be noted that the study was supported by the Welcome Trust and that the lead author, as well as one additional author, work for the Welcome Trust, a pharmaceutical giant that makes multibillion dollar revenues from AIDS medications like Combivir, Trizivir and Retrovir (AZT, Azidothymidine). These researchers-involved in conflicts of interest-will hardly be able to say that HN has not been proven to exist.

- Of 75 particles, the paper said that five had no well-defined core, had a single core, three had a complete core plus part of a further core, while four particles had two cores; the particles with two cores were larger than those with only one. For one thing, one notices that no double-cores can be seen in the printed pictures, writes Canadian biologist and AIDS expert David Crowe, and for another, the question arises: how can a virus have two cores at all? That would be something absolutely new!

- In the majority of single-cored particles, the core was cone-shaped (morphology); in the remaining particles, on the other hand, the cores were tube-shaped (cylindrical), triangular or simply shapeless. Here as well, it is difficult to comprehend that all these particles with such different appearances could all belong to a very particular type of retrovirus (for that is what HN is supposed to be).

- Particles were of a great variety of sizes: the diameters measured by Briggs et al ranged from 106 - 183 nanometer (one billionth of a meter). Is it advisable to classify all the particles as being one and the same particular type? People, for example, vary in size. Let’s say we were comparing men and assumed that the average man is 1.78 meters or 5.84 feet tall. If the margin measured by Briggs et al (106 - 183 nanometers) were carried over, we would get heights ranging between 1.30 and 2.25 meters (4.27 and 7.38 feet). This would hardly permit us to believe that we were dealing exclusively with full-grown males. It also speaks against the assumption that the particles of such various sizes, which originate from one cell culture, are all of the same virus type.

- AIDS researcher Val Turner of the Australian Perth Group re-measured the diameters of the particles that were visible in diagram 1A of Briggs et al’s paper. This revealed that two of the particles (also called virions, which gives the impression that they belong to a virus that had invaded from outside) had diameters of even less than 100 nanometers.

- The Structure articles authors themselves conceded that both printed images (which originated from one image) are not representative of the entire sample, but that begs the question: what shapes and sizes are the particles in the pictures that were not shown? This information was not provided even when requested.

- In this context, according to relevant sources, the diameter of retrovirus particles (HN is
supposed to be a retrovirus, after all) are quoted as 100 - 120 nanometers, something that clearly deviates from the 106 - 183 nanometers measured by Briggs.

- It would have cleared up a lot in this context if scientists had undertaken a complete purification and characterization of the particles, as David Crowe remarks, but this apparently did not happen. The researchers themselves say that only particles with minimal contamination were available.

- Not once is a virus purification method described in the Structure paper; in this regard, lets refer to an article by Welker et al, published in the Journal of Virology in 2000. They first say, remarkably, that, it is important to have pure HN particles available, which confirms how important virus purification is for virus detection. However, they did not demonstrate that pure HN had been extracted; it was also said the electron microscopic analysis showed that the core preparations were not completely pure.

- And even if the particles were pure, the problem still arises that even after the purification process, cell components (known as microvesicles, micro bubbles, and material of cellular origin) could be present, which even from an orthodox perspective are non-viral, although they may have the same size and density as so-called HN. Thus we read in a paper published in the journal Virology: Identification and quantization of cellular proteins associated with HN-1 particles are complicated by the presence of non-virion-associated cellular proteins that co-purify with virions.

HIV = AIDS?
Is HN the cause of AIDS? Lets allow the medical establishment speak for itself. Reinhard Kurth, director of the Robert Koch Institute (one of the pillars of mainstream AIDS research), conceded in Der Spiegel (9 September, 2004): We don’t exactly know how HN causes disease. In the 1996 documentary AIDS-The Doubt, by French journalist Djamel Tahi (broad casted on German Arte Television), Montagnier admitted to the same, saying, there is no scientific proof that HN causes AIDS. And 12 years before, in 1984, Montagnier emphasized that, the only way to prove that HN causes AIDS is to show this on an animal model. But there is still no such model. The California Monthly, the UC Berkeley alumni magazine, confronted Nobel laureate Kary Mullis in an interview using a statement from another Nobelist, David Baltimore. [Dear Mr. Mullis,] you mentioned Baltimore a moment ago. In a recent issue of Nature, he said : There is no question at all that HN is the cause of AIDS. Anyone who gets up publicly and says the opposite is encouraging people to risk their lives.

Whereupon Mullis replied : I’m not a lifeguard, I’m a scientist. And I get up and say exactly what I think. I’m not going to change the facts around because I believe in something and feel like manipulating somebody’s behavior by stretching what I really know. I think it’s always the right thing and the safe thing for a scientist to speak one’s mind from the facts. If you can’t figure out why you believe something, then you’d better make it clear that you’re speaking as a religious person.

People keep asking me, You mean you don’t believe that HN causes AIDS? And I say, Whether I believe it or not is irrelevant! I have no scientific evidence for it! I might believe in God, and
He could have told me in a dream that HN causes AIDS. But I wouldn’t stand up in front of scientists and say, I believe HN causes AIDS because God told me. I’d say, I have papers here in hand and experiments that have been done that can be demonstrated to others. It’s not what somebody believes, its experimental proof that counts. And those guys [from AIDS orthodoxy] don’t have that.

The most significant diagnostic tools of viral and AIDS medicine are:
1. Antibody tests (HN tests)
2. PCR viral load tests
3. Helper cell counts (T-cells, or rather the T-cell subgroup CD4)

These are what is known as surrogate markers: alternative methods which doctors determine, on the basis of laboratory data, if someone is infected with HIV or not, and whether they have AIDS. Instead of using traditional methods for investigating whether real disease symptoms (so-called clinical endpoints) have occurred, AIDS doctors look at whether the number of CD4 cells has decreased within a certain time period; if so, the risk of contracting AIDS is said to be low.

But as previously mentioned the results given by these methods are highly dubious ways to detect viruses like HIV, the SARS corona virus, or the avian flu virus HSN1 and their pathogenic effects. Often enough, surrogate markers have led to misdiagnosis.

Let’s look first at the HIV antibody tests. They’re based on an antigen-antibody theory, which assumes the immune system fights against these antigens (proteins from HIV), as they are called, which are seen by the body as foreign. Their detection triggers an immune reaction, or response, which in turn induces the formation of specifically targeted antibodies.

Now, since these so-called HIV antibody tests only prove the existence of antibodies (and not, it is worth noting, the antigen directly, which in this case would be parts of HIV), we have to assume that HIV must have been detected during the validation of the tests. Only then could one use the antigen to calibrate the antibody tests for this particular (HIV) antigen. That is, only in this way can one test whether HIV antibodies are present or not, and, if HIV has not been proven to exist, the tests cannot possibly be known definitively to react to it.

When you know this information, the antibody test manufacturers insert isn’t quite so surprising. It dearly states there is no recognized standard for establishing the presence or absence of antibodies to HIV-1 and HIV-2 in human blood. Reacting to this interesting fact, and in reference to a paper by the Australian Perth Group (published in the scientific journal Nature Biotechnology) the German weekly newspaper Die Woche ran a headline calling it, The AIDS Test Lottery. The article went on to say that the antibody tests do not measure what they should: HIV infection. They also react to people who have overcome a tuberculosis infection. [Yet] the world’s leading AIDS researchers at the Institute Pasteur in Paris reviewed the study before publication.

But what do the tests react to, then, if not to HIV? As we’ve already noted with AIDS, a circular definition has also been used with the antibody tests: in the mid-1980s, the proteins which caused the tests to react most strongly were selected from blood samples from seriously ill AIDS patients, and used to calibrate the tests.
That these proteins have something to do with HIV, or at least are similar to a retrovirus of whatever type, has, however, never been proven. And, in fact, antibody tests were not actually designed specially to detect HN at all, as Thomas Zuck, of the American drug approval authority FDA, warned in 1986. Rather, blood tests should be screened for their resistance to false-positive reactions due to other germs or contaminants (something which also fits with what Die Woche wrote: that HN tests also reacted in people who had survived tuberculosis; and also dozens of other symptoms, including pregnancy or simple flu, could cause a positive reaction).

But to stop using these HIV tests was simply not practical, as Zuck admitted at a World Health Organization meeting. Now that the medical community had identified HN as an infectious sexually transmitted virus, public pressure for an HN test was just too strong.

With HN antibody tests, orthodox AIDS research turned traditional immunology upside-down, by informing people who had positive antibody tests that they were suffering from a deadly disease. Normally, a high antibody level indicates that a person had already successfully battled against an infectious agent and is now protected from this disease. And since no HN can be found in AIDS patients, the hunt for a vaccine is also an irrational undertaking. Even Reinhard Kurth, director of the Robert Koch Institute made a sobering comment in the Spiegel in 2004: To tell the truth, we really don’t know exactly what has to happen in a vaccine so that it protects from AIDS.

Viral load measurements with the help of the polymerase chain reaction (PCR) are just as dubious and ultimately meaningless. As long as HN has not been proven to exist, these tests cannot be calibrated for HN-and they cannot be used to measure HN viral load. Very fine traces of genes (DNA, RNA) may be detected, but whether they come from a (certain) virus, or from some other contamination, remains unclear.

Heinz Ludwig Sanger, professor of molecular biology and 1978 winner of the renowned Robert Koch Prize stated that HN has never been isolated, for which reason its nucleic acids cannot be used in PCR virus load tests as the standard for giving evidence of HN. Not coincidentally, relevant studies also confirm that PCR tests are worthless in AIDS diagnosis: for example, Misdiagnosis of HN infections by HN-1 viral load testing: a case series, a 1994 paper published in the Annals of Internal Medicine.

In 2006, a study published in the Journal of the American Medical Association (JAMA) shook again the foundation of the past decade of AIDS science right to the core, inciting skepticism and anger among many HN = AIDS advocates. A US nationwide team of orthodox AIDS researchers led by doctors Benigno Rodriguez and Michael Lederman of Case Western Reserve University in Cleveland disputed the value of viral load tests—the standard used since 1996 to assess the patients health, predict progression to disease, and grant approval to new AIDS drugs—after their study of 2,800 positively tested people concluded viral load measures failed, in more than 90% of cases, to predict or explain immune status.

While orthodox AIDS scientists and others protest or downplay the significance of the JAMA article, Rodriguez’s group stands by its conclusion that viral load is only able to predict
progression to disease in 4% to 6% of (so-called) HIV positives studied, challenging much of the basis for current AIDS science and treatment policy.

The same controversy plagues tests that count CD4 helper cells. Not a single study confirms the most important principle of the HIV = AIDS theory: that HIV destroys CD4 cells by means of an infection. Furthermore, even the most significant of all AIDS studies, the 1994 Concorde study, questions using helper cell counts as a diagnostic method for AIDS—and many studies corroborate this. One of these is the 1996 paper Surrogate Endpoints in Clinical Studies: Are We Being Misled? Printed in the Annals of Internal Medicine, the paper casually concludes that CD4 count in the HIV setting is as uninformative as a toss of a coin—in other words, not at all.

Following the news that viral load is not an accurate method of assessing or predicting immune status comes word from the Journal of Infectious Diseases that helper cell counts may be less reliable measures of immune competence than the AIDS orthodoxy previously believed. The study conducted in Africa by the World Health Organization (WHO) revealed that so-called HIV negative populations can have T-cell counts below 350, a number that would, according to WHO guidelines, qualify for an AIDS diagnosis in HIV positive populations. Another surprising conclusion (from the point of view of the HIV = AIDS believers) from the same WHO study: HIV positives that started AIDS drug treatment with low helper cell counts had the same survival outcomes as HIV positives that began treatment with high T-cell counts!

One of the most spiteful and most un-healing properties of scientific models is their capability to strike down truth and take its place, warns Erwin Chargaff, longtime professor at Columbia University’s Biochemical Institute in New York. And often, these models serve as blinkers, by limiting attention to an excessively narrow area. The exaggerated trust in models has contributed much to the affected and in genuine character of large parts of current natural research.

The biotechnology company Serono illustrates the ways in which such surrogate marker tests can be misused. The Swiss firm was suffering revenue losses with their preparation Serostim, which is supposed to counteract the weight-loss so typical of AIDS patients. So, at the end of the 1990s, Serono redefined this AIDS wasting and developed a computerized medical test, which would professedly determine body cell mass. These tests were actually adopted by doctors. And so it came about that doctors ordered Serostim when the tests showed patients had lost body cell mass, a treatment that could easily cost more than $20,000. The strange thing was that patients who, with the help of the tests, had been diagnosed with a reduced body cell mass, had in reality not lost any weight at all. On the contrary, some had even gained weight. The Serostim scheme was finally busted and, as a legal investigation showed, more than 80% of Serostim prescriptions had been unnecessarily ordered through the tests application. Michael Sullivan, the attorney in charge of the investigation, termed the tests voodoo magic, and they ultimately cost Serono more than $700 million in criminal fines. At that point, this was the third highest sum ever to be paid in such a judicial process.

There is much evidence that AIDS—that conglomerate of dozens of well known diseases—can substantially be explained by the intake of poisonous drugs and medications (antivirals, antibiotics, etc.) and by malnutrition. Around 80% of all children declared to be AIDS patients
are born to mothers who have taken intravenous drugs that destroy the immune system. And the first people to be diagnosed as AIDS patients in the USA were all consumers of drugs like poppers, cocaine, LSD, heroin, ecstasy, or amphetamines, all of which have devastating effects on the immune system. The American National Institute on Drug Abuse was not alone in confirming the extreme toxicity and immunosuppressive effects of substances like heroin or poppers (nitrite inhalants) used among gay men.

With poppers, the following chemical event takes place: poppers are nitrites, and when inhaled are immediately converted into nitric oxide. Through this, the blood's capability to transport oxygen is compromised; it oxidizes. The first areas to sustain damages through this oxygen deficiency are the linings of the smallest vessels (epithelia). When this damage develops malignantly it is called Kaposi sarcoma—a vascular tumor that is diagnosed in many AIDS patients. And, as a matter of fact, tumor tissue is oxidized.

This self-destructive process is particularly noticeable in the lungs, since poppers are inhaled and dead organic material is produced, which cannot be completely disposed of by the cells weakened detoxification systems. At this point, fungi enter the game. Nature intended precisely this role for them because they eat away all kinds of waste. This explains why so many patients, termed AIDS cases, suffer from pneumocystis carinii pneumonia (PCP), a lung disease typically associated with strong fungal infestation (decay).

These patients’ immune systems are weakened, which is the common denominator for the development of PCP, according to Tinstey Harrison’s textbook for internal medicine. And the disease [the immune deficiency upon which PCP develops] can be produced in laboratory rats by starvation or by treatment with either corticosteroids [cortisone] or cyclophosphamides. In other words, with cell-inhibiting substances that are destructive to the immune system, just like AIDS therapeutics. This makes it obvious that there is no need for HN to explain AIDS (which is nothing but a synonym for well-known diseases like Kaposi sarcoma or PCP).

Correspondingly, the typical sufferer who is tagged as an AIDS patient suffers from malnutrition; particularly those affected in poor countries, but also many drug users who constitute the bulk of AIDS patients in wealthy countries. At the same time, studies show that a stress factor like drugs can trigger a new arrangement of genetic sequences (DNA) in the cells, whereby cell particles are formed-particles produced (endogenously) by the cells themselves (and interpreted by the medical industry as viruses invading from the outside, without any proof)

Five severely ill homosexual young men became the first characters in the AIDS story, in 1981. American scientist Michael Gottlieb, from the Medical Center of the University of California in Los Angeles, had brought these five patients together after a search of several months, using the highly dubious clustering method. Gottlieb dreamed about going down in the history books as the discoverer of a new disease. The afflicted patients suffered from the pulmonary disease pneumocystis carinii pneumonia (PCP). This was remarkable, because young men in their prime years do not usually suffer from this, but rather babies who come into the world with an immune defect, older adults, or those on immunosuppressive medication (which burdens or damages the immune system).

The medical researchers apparently took no other factors into account concerning the causes, as
the patients drug use. Instead, the medical establishment and above all the Center for Disease Control (CDC) gave the impression that the cause of PCP was completely mystifying, so the basis was set to launch a new disease. The CDC eagerly seized up Gottliebs theses: Hot stuff, hot stuff, cheered the CDCs James Curran. It was so hot, that, on 5 June 1981, the CDC heralded it as a red-hot piece of news in their weekly bulletin, the Morbidity and Mortality Weekly Report (MMWR), which is also a preferred information source for the media.

In this MMWR, it was immediately conjectured that the puzzling new disease could have been caused by sexual contact, and was thus infectious. In fact, there was no evidence at all for such speculation, for the patients neither knew each other, nor had common sexual contacts or acquaintances, nor had they comparable histories of sexually transmitted diseases.

Sex, being three billion years old, is not specific to any one group-and thus naturally does not come into question as a possible explanation for a new sort of disease, points out microbiologist Peter Duesberg of the University of California, Berkeley. But buried in Gottliebs paper was another common risk factor [criminally neglected by the CDC] that linked the five patients much more than specifically than sex. These risk factors included a highly toxic lifestyle and use of recreational drugs that were massively consumed in the gay scene, primarily poppers, or in medical jargon nitrite inhalants.

Inhalants is used because these drugs are normally sniffed from a small bottle, and like the customary poppers expression the term can be traced back to the mid 19h century. In 1859, the vasodilatory effect that follows inhalation of amyl nitrite was described. This led to its first therapeutic use in 1867 as muscle relaxants for (cardiac disease) patients suffering from angina pectoris (chest pain). The original form of the drug was glass ampules enclosed in mesh: they were called pearls. When crushed between the fingers, they made a popping sound; hence, the colloquialism poppers evolved.

The US National Institute on Drug Abuse (NIDA) dates their use as recreational drugs from 1963. From then on, the drug experienced a proper boom, assisted by the fact that in industrialized countries like the USA, drug consumption in general sharply increased in and since the 1960s and 1970s, the years of sexual and political revolution (between 1981 and 1993, alone the number of cocaine overdose victims delivered to hospitals jumped from 3,000 to 120,000, a 4,000% increase).

The gay scene made use of poppers well-known muscle relaxant property. Taking poppers enables the passive partner in anal intercourse to relax the anal musculature and thereby facilitate the introduction of the penis, according to a 1975 report in the journal Medical Aspects of Human Sexuality. Poppers also helped prolong erection and orgasm. The substance was (and is) easy to make at home, and it is very cheap to buy (a few dollars per vial). At the same time, poppers were massively advertised in popular gay media. And for promotional purposes, the drugs even had their own comic strip spokesperson-a handsome blond hunk who promoted the (in truth, irrational) idea that poppers make you strong and that every homosexual simply had to take them.

NIDA reported that sales of in just one US state added up to $50 million in 1976 (at $3 per vial
that equals more than 16 million bottles). By 1977, poppers had permeated every angle of gay life, writes Harry Haverkos, who joined the CDC in 1981 and the American drug authorities NIDA in 1984 and was the leading AIDS official for both institutions. And in 1979, more than five million people consumed poppers more than once a week.

Poppers can severely damage the immune system, genes, lungs, liver, heart, or the brain; they can produce neural damage similar to that of multiple sclerosis, can have carcinogenic effects, and can lead to sudden sniffing death. Even the drugs label warns it is highly flammable; may be fatal if swallowed. And the medical establishment knew about its various dangers. In the 1970s, the first popper warnings appeared in scientific literature. In 1978, for instance, L.T. Sigell wrote in the American Journal of Psychiatry that the inhaled nitrites produced nitrosamine, known for its carcinogenic effects-a warning which Thomas Haley of the Food and Drug Administration (FDA) likewise articulated.

In 1981, the New England Journal of Medicine (NEJM), one of the world’s most significant medical journals, published several articles at the same time singling out the so-called fast-lane lifestyle as a possible cause of AIDS. This lifestyle is characterized by an extremely poor diet and long-term intake of antibiotics and antifungal substances, which damage the mitochondria, the cells powerhouses (plus numerous other medicines, later primarily chemotherapy-like antiviral AIDS preparations including AZT, ddC, d4T, aciclovir and ganciclovir).

Besides poppers, many other, likewise highly toxic, drugs were on the menu, including crystal meth (methamphetamine), cocaine, crack, barbiturates, ecstasy (XTC), heroin, librium, LSD, mandrex, MDA, MOM, mescaline, mushrooms, purple haze, Seconal, special K, tuinol, THC, PCP, STP, DMT, LDK, WOW, window pane, blotter, orange, sunshine, sweet pea, sky blue, Christmas tree, dtist, Benzedrine, Dexedrine, Dexamyl, Desoxyn, clogidal, nesperan, tytch, nestex, black beauty, certyn, preludin with B 12, zayl, quaalude, tuinal, Nembutal, amytal, phenobarbital, elavil, valiurn, darvon, mandrax, opium, stidyl, halidax, caldfyn, optimil, and drayl.

David Durack asked the (still relevant) question in his lead article in the December 1981 NEJM: how can AIDS be so evidently new, when viruses and homosexuality are as old as history? lifestyle drugs, according to Durack, should be considered as causes. So-called recreational drugs are one possibility. They are widely used in the large cities where most of these cases have occurred. Perhaps one or more of these recreational drugs is an immunosuppressive agent. The leading candidates are the nitrites [nitrite inhalants, poppers], which are now commonly inhaled to intensify orgasm.

American author and AIDS chronicler Randy Shilts addresses this issue in his famous 1987 work The Band Played On: [The poppers-AIDS starting point] would explain why the disease appeared limited to just three cities—to New York, Los Angeles and San Francisco, the three centers of the gay community, a conspicuous feature also described in the CDCs MMWR from 24 September, 1982.

Durack additionally notes that, other than drug-using homosexuals, the only patients with AIDS symptoms were junkies. In fact, in affluent nations like the USA or Germany, intravenous drug
users have always made up a third of all AIDS patients, a fact that hasn’t been acknowledged to the general public.

Immune system destruction is even more common among intravenous drug users than poppers-inhaling homosexuals. Junkies lives are wrecked not by a virus, but (primarily) by excessive drug use over years. If the general public had known that a consistently high percentage of AIDS patients were intravenous drug addicts, perhaps the medical establishment would have been forced to study drugs as a possible cause of AIDS.

A number of high-power organizations sought to prevent this message from getting through. First, the CDC purposely skewed their statistics. Their weekly bulletins divided AIDS patients into groups (homosexuals, intravenous drug users, racial minorities, hemophiliacs), yet they attributed a lower percentage to junkies than homosexuals. At one point, 17% were identified as drug users, and 73% were homosexuals, according to the CDC. This gave the impression that drug users were a less significant group among AIDS patients.

The CDC only admitted they played with the numbers to those who meticulously probed for more information. Journalist and Harvard-educated analyst John Lauritsen discovered that 25% of AIDS patients statistically labeled homosexual were also drug users. But the CDC simply lumped all of these gay drug addicts into the homosexual category. For this reason, the portion of drug users was 17% whereas in reality it should have been 35% (that is, more than one in three AIDS patients fits into the intravenous drug user category).

Based at least in part on these skewed stats, the gay community certainly became active in the AIDS war and some became powerful gatekeepers of the AIDS establishment. Gay men, some of them affluent and relatively privileged, found their way into private doctors offices and prominent teaching hospitals—and from there into the pages of medical journals [and from there into the mass media]—while drug users often sickened and dies with little fanfare, describes sociologist Steven Epstein. And many reports in medical journals were penned by doctors who were very close to the gay scene and for that reason had treated many AIDS patients.

The focus on homosexuals was so strong that, at the beginning AIDS was even called Gay-Related Immune Deficiency Syndrome (GRID). Or simply, gay disease, primarily because clinicians, epidemiologists, and reporters perceived [the syndrome] through that filter of the gay men’s health crisis, as Epstein outlines.

It was also far from random that the first Spiegel cover on AIDS depicted two well-endowed young men, looking at each other’s genitals (see picture). But with gays, focus remained on the topic of sexual transmission, and drug use was not tied in. And so it was also said right at the beginning of the first Spiegel cover story in 1983: An Epidemic That Is Just Beginning: the gay epidemic, AIDS, a deadly immune deficiency, has reached Europe.

These media messages quickly caused widespread belief and panic that a deadly contagious sexually transmitted epidemic was occurring, at least among gay men. Even though there was no scientific data to back these perceptions up and Gallo and Montagnier had yet to publish their 1984 papers, claiming to have discovered HN as the cause of AIDS.
Why was the gay scene such a focus of interest? And the much more obvious connection between drugs and immune disorders ignored? Particularly since in developed countries, almost all patients said to have the one of the immune deficiency diseases called AIDS have always been homosexuals and drug users. In other words, almost all AIDS patients take immune-suppressive and potentially deadly drugs and/or medications.

Firstly, mainstream culture knew next to nothing about poppers and they are still used almost exclusively in the gay community. In the 1980s, gay organizations strongly objected to the idea that their much-loved drugs could play a role, particularly a decisive role, in the development of AIDS symptoms. The AIDS establishment, attached to its virus-fixation, also lured the community into their fold by creating opulently paid consulting contracts for important members of gay organizations. Pharmaceutical companies also invested money in the gay community with innumerable advertisements for AIDS medications, like a Hoffmann-La Roche ad reading, Success creates courage, and a Welcome ad for poppers calling amyl nitrite [i.e. poppers] the real thing.

The gay community even ignored urgent medical warnings from scientists about the dangers of poppers. Editors of The Advocate, a popular US magazine for homosexuals, ignored their letters, but accepted a whole series of poppers advertisements called Blueprint for Health from Great Lakes Products, at the time probably the largest manufacturer of sex drugs. In this, it wrongly said that government studies had exonerated poppers from any connection to AIDS, and that poppers were harmless, writes John Lauritsen, who has studied the topic of poppers and AIDS in depth.

These ads also suggested that poppers-just like vitamins, fresh air, exercise and sunshine-belonged to a healthy lifestyle, and that they were an integral part of the gay community’s Fantasyland and wonderful land of drugs, parties and sex.

Chapter 3
The scene is no different today. Although certain versions of the drugs were prohibited because of high toxicity in 1988 and 1990, promotional websites for the lifestyle drug, such as bearcityweb.com or allaboutpoppers.com claimed that poppers are the closest thing to a true aphrodisiac that exists today, and in addition they have been shown to be among the safest and most pleasurable compounds the world has ever seen.

Many important gay publications and organizations continue to promote poppers and censor data on adverse effects. This has had devastating consequences in society, since the gay media play an important role in informing and educating writers and journalists, who themselves deliver important messages about AIDS to the general public. Indeed, some media organs of the AIDS movement, such as AIDS Treatment News, are widely recognized as agenda-setting vehicles for the circulation of scientific knowledge, and are read by activists, doctors, and researchers alike, writes Steven Epstein.

A further decisive building block on the way to the construction of the dogma that AIDS is a contagious viral disease was the behavior of the Centers for Disease Control (CDC). From the beginning, they were unwilling to explore the drug connection. The CDC set on the search for a
deadly virus, without hesitating to suppress disagreeable data. In 1982, their own AIDS expert Haverkos analyzed three surveys of AIDS patients conducted by the CDC. He came to the conclusion that drugs like poppers did play a weighty role in disease onset.

But the CDC refused to publish their own high-ranking employees study, and Haverkos transferred to the FDA in 1984 to become AIDS coordinator there. The paper finally appeared in the journal Sexually Transmitted Diseases in 1985. This prompted the Wall Street Journal to pen an article unambiguously stating that drug abuse was so universal among AIDS patients that this, and not the virus, must be considered the primary cause of AIDS.

But such reports fell on deaf ears, for the world had already been sent down the virus road years before. Talk of drug factors ended with the CDCs second AIDS related MMWR (3 July, 1981), in which further highly unusual cases of Kaposis sarcoma were reponed. This had a viral effect upon media coverage. When the first reports of the peculiar deadly illness from California began to wash up here, the CDC releases were our only proper source of information, remembers Hans Halter, who penned the Spiegel’s first cover story on AIDS. Its headline: An epidemic that is just beginning.

Halter, himself a specialist in sexually transmitted diseases, had, as he relates, looked through the CDC data with a virologist friend. It was clear to us, Halter claims that a retrovirus transmitted through sperm and blood was to blame!

Halter admitted in that story that the immune system [in homosexuals], as scientific examinations show, is also compromised through antibiotic treatment, drug consumption, and intensive use of poppers.

Yet, incomprehensibly, in the very same article, only a few paragraphs previously, Halter wrote: First, the poppers hypothesis collapsed: a control group of non-AIDS-infected homosexuals also took the stimulant, which expands blood vessels and is said to improve orgasm. Not only does this contradict Halter’s own understanding that a drug lifestyle damages the immune system. Also, even if the experiment Halter mentioned had actually existed, this is still a far cry from demolishing the hypothesis that poppers play a (significant) role in the onset of the disease symptoms termed AIDS.

You would think this writer must have first reviewed this study to come to such a conclusion. What exactly was being investigated? Was the paper compiled without bias or conflicts of interest? Is the argument conclusive? We don’t know because no such study has ever been conducted. It’s no wonder that Halter couldn’t name the study upon request. Instead, he recommend d looking in Shilts book, And the Band Played On, adding, maybe there are answers in it Indeed there are. According to Shilts, the poppers starting-point does offer an explanation for AIDS. Everybody who got diseases seemed to snort poppers, writes Shilts.

Of course, there will always be people who take drugs like poppers and do not get one of the AIDS diseases like lymphatic cancer. But dosage and the length of time a person uses a drug, as well as other individual behavior patterns, living conditions, and genetic make-up always play a role. Just as a casual smoker is less likely to get lung cancer than a chronic smoker.
On 11 February 2005, Dr. Thomas Frieden, a New York City health official, stepped up to the microphone and announced the discovery of a supposedly deadly new strain of HIV that was resistant to around 20 different AIDS medications. The world press went ballistic. German newspaper Die Welt headlined: Super-AIDS in New York, and the Süddeutsche Zeitung speculated that the one gay male whose illness had led to Dr. Frieden’s big announcement had become infected with the virus at a bareback party, a gay sex party (bareback refers to anal sex without a condom). It was only incidentally mentioned in the article that the man had taken drugs including cocaine and crystal meth (methamphetamines) to keep him going all night long.

By the end of the month, an article in the gay/lesbian magazine San Francisco Bay Times, points out that, what the [mainstream] media has failed to report is that the 46-year-old patient had been on a three-month run of crystal [meth], 90 days in a row, [and] when he [finally] went to the doctor, he was just a shell of a person.

The man had also been a chronic drug-taker since the age of 13: first marijuana and alcohol, then later heavier drugs like cocaine or crystal meth-substances that have similarly stimulating and short-term performance-enhancing effects, and are just as toxic as poppers (which were probably also among the drug-repertoire for the man in his mid-40s).

We are looking at an example of a classic AIDS patient. Let’s remember here that the first AIDS patients were described as young homosexuals heavily addicted to drugs, ranging in age from 30 to mid-40. How then, could these patients possibly be helped by further chemical poisoning in the form of highly toxic medications? That the above-mentioned patient did not respond positively to any of the twenty AIDS medications had nothing to do with a drug-resistant virus (as is continually asserted), but rather to the fact that the already unhealthy, immune-compromised man could not handle the highly toxic preparations.

Shortly after the news of a mutant HIV strain, a striking article appeared in Science, acknowledging that there was still no proof that what had been termed the nightmare virus strain can cause disease. Jacques Normand, director of AIDS research at the US National Institute on Drug Abuse (NIDA), confirmed in an interview we got published in the weekly newspaper Freitag, that the question of whether we are dealing with a super AIDS virus remains unanswered. And drugs, continued Normand, cannot be ruled out as the main cause of the 46-year olds health problems.

These sentences carry even more weight when you consider that both the drug administration and specialist journals like Science normally stay right in line with orthodox AIDS medicine, and that real criticism or doubts on the HIV = AIDS dogma are rarely ever heard.

At a high-level meeting of US health authorities in 1994--titled Do Nitrites Act as a Co-Factor in Kaposis Sarcoma?--The best-known speaker was the National Cancer Intitutes Robert Gallo, so-called co-discoverer of HIV. His statements were noteworthy. According to Gallo, HIV was surely a catalytic factor in Kaposis, but even he acknowledged, there must be something else involved. Then he added: I don’t know if I made this point clear, but I think that everybody here knows-we never found HIV DNA in tumor cells of KS. So this is not directly transforming. And in fact we’ve never found HIV DNA in T cells although we’ve only looked at a few. So, in other words we’ve never seen the role of HIV as a transforming virus in any way.
And in response to a question from Harry Haverkos, then director of the AIDS department at NIDA, who said that not a single case of KS had been reported among blood recipients where the donor had KS, Gallo allowed: The nitrites [poppers] could be the primary factor.

To fully appreciate Gallo’s statement, we must recall that, in wealthy nations like the USA and Germany, Kaposi’s sarcoma was—next to PCP—the most significant disease among patients labeled with AIDS. In 1987, for example, Der Spiegel described Kaposi’s sarcoma patients defined as AIDS patients as the sarcoma covered skeletons from the same-sex scene.

Indeed, At present, it is accepted [even by CDC scientists] that HIV plays no role, either directly or indirectly, in the causation of Kaposi’s sarcoma, writes Australian medical professor and AIDS expert Eleni Papadopulos. Given this background, it seems paradoxical that Kaposi’s sarcoma is still part of the official AIDS definition in industrialized countries (anyone with KS and a positive test result counts as an AIDS patient)—and that, contrary to the facts, even respected magazines like The New Yorker still assert that Kaposi’s sarcoma is a sign of AIDS (i.e. HIV causes KS).

The media tend to have difficulties with the facts anyway. They prefer to occupy themselves with their favorite theme: sex. By the end of 1982, dozens of articles on the mysterious new disease had appeared in the US print media alone. Soon enough, the number jumped to hundreds per month. And they constantly tossed around the idea that this virally-caused and sexually transmitted disease posed a threat to the general public. In Germany, the news magazine Der Spiegel took a leading role in this virus propaganda, publishing approximately 20 cover stories on HIV/AIDS since 1983, and, according to a Spiegel’s internal release, the magazine has reported far more on AIDS than on any other medical topic, including cancer.

By late 1984, the Hamburg-based news magazine was so confident with its AIDS dossier that they headlined, The Bomb Is Planted and that, in developed nations like Germany the epidemic is breaking out of the gay-ghetto. Women are also in danger. The following year, Der Spiegel explicitly expressed certainty that everyone was at risk with the cover story headline: Promiscuity Is the Epidemics Motor. The story goes on to state it has become clear that the disease has started to reach out from its previous high-risk groups [homosexuals and intravenous drug users].

The article went on to offer up the doctor’s orders for curbing the spread of HIV: Still without a cure in the fight against AIDS, doctors advise monogamy to heterosexuals and celibacy to gays. To support these theses, the magazine, which in Germany still epitomizes investigative journalism, looked to headlines from the rainbow press, including Danger For Us All: A New Peoples Epidemic from The Munich glossy Quick and AIDS-Now the Women Are Dying from the master of media warhorses, the Bild am Sonntag.

The Spiegel practiced a juicy double strategy by incorporating the tabloid medias sensationalized statements into its text in such a way that they substantiated The Spiegel’s own theses. Yet it tried to distinguish itself from the cheap tabloids by writing that hardly a day goes by without the boulevard press seizing up the subject [of AIDS] with headlines that go down easy. But Der Spiegel was fully invested in the game of muckraking AIDS coverage.
Particularly in the 1980s, Spiegel had sex on the brain, so articles were teeming with questions like, Should only homosexuals believe in it, maybe because the Lord has always had a whip waiting for them? So-called journalists gushed about doing it upright and cock-centered routines and lamented the end of the quickie or the good old one-night stand. And where would tabloid journalism be without reporting on Hollywood stars fears of AIDS? According to Der Spiegel, Linda Evans, who was thoughtlessly kissed by AIDS-infected Rock Hudson from the Denver Clan, awoke night after night in terror. She cries on the telephone for help, for her nightmares show her all the stages of the disease. Burt Reynolds has to reaffirm again and again that he is neither gay, nor has AIDS. Or what about this hook? Rock-Vamp Madonna and other pop stars back off singing: Take your hands off me.

Bo Derek, the sex icon of the 1970s and 1980s, was even forbidden [by her husband] to kiss on-the-job, except with AIDS-tested film stars, according to the Credo: No kiss, no AIDS. All sorts of celebrities weighed in with their own brand of homophobic hysterics, like Denver Clan star Catherine Oxenberg, who said, If I have to work with a gay in the future, I won’t kiss him. Der Spiegel even took a jab at the US President: 30% of all actors are gay. Does Ronald Reagan know that? Rock Hudson seemed to be the prime target of every AIDS-related riff: The beasts with AIDS threaten Hollywood society. To counter the hysteria, Ed Asner, the esteemed president of the Screen Actors Guild, suggested striking kissing scenes from screenplays for the time being. Now it’s getting serious, by holy [Rock] Hudson!

Kissing phobia became so infectious that the CDC issued an official notice that Kissing is not a risk factor for the transmission of AIDS. In his 1987 cover story, Spiegel writer Wilhelm Bittorf didn’t shy away from giving his own personal views, portraying the homosexual community as a potential pest hole, and sexual interaction with a single woman as a necessary evil: A woman who I had slept with a few times, and who I found rather exciting, later told me that she was particularly proud that she had also converted gays to her charms. Gays! I felt as if someone had rammed a giant icicle into my gut. The fear that I had gotten myself infected was enormous. I have no idea why. Of course, I had earlier read, and written, a lot about AIDS, but the fear first clutched me there. The weeks leading up to the decision to take the blood test were awful. It is as if you submit yourself to an irrevocable judgment of your entire life. Then the blood test, anonymous; a week of waiting, hardly sleeping at night: one can only think of oneself. Test result: negative. But the shock is still bone deep. My sex life according to the motto good is what turns you on has been over since that time. Sex afterwards, unlike beforehand, was sex with a condom, even when the girls grumbled about it. And now, months of living with just one, who I chose based on the criteria of whether she can be faithful. I live monogamously and am concentrated on just one person. I do lust after others, but I deny myself.

That the Spiegel readers do not know more, as the magazine is fond of saying about itself in its ads, becomes clear when one looks more closely at coverage since the beginning of the 1990s. Since then, Der Spiegel has forced the constant interplay between fanning hopes and dashing them, continually stringing its readers along emotionally. In the 1991 story Mother Nature Improved, AIDS pioneer Robert Gallo was quoted, boasting: In ten years at most, a vaccine against AIDS will have been developed and will be ready to use; and in 1995, it was optimistically reported that after the disappointment with AZT, the new pill of hope from Basel is being generated by the kilogram in the cauldrons of the Swiss group Heffernan-La Roche:
Then in 1996, sudden pessimism: Since 1985, virologists, epidemic doctors, geneticists, and pharmaceutical researchers have discussed the pandemics fatal march of victory at international AIDS congresses. The sobering result was constantly the same: AIDS can apparently not be brought under control, possibility of a cure or an effective vaccine still lies in the distant future.

Only one year later, when the pharmaceutical industry brought new active substances onto the market, Der Spiegel conveyed to its readers, another uplifting message: Now, words of hope are everywhere-Newsweek and the New York Times proclaim a possible end of AIDS.

Yet were still no closer to the end of AIDS. This did not escape the Spiegel either; the magazine quoted Reinhard Kurth, director of the Robert Koch Institute, with these resigned words: The optimism of the beginning of the 1980s is long gone, since vaccines limiting the transmission of AIDS are the only way that promises long-term success against this most serious medical catastrophe of modern times; [but] , the simplest roads to the development of an HN vaccine are unfortunately blocked.

To this, media researcher Michael Tracey writes that media coverage of AIDS satisfied a certain kind of news value that is ignorant but loves to wallow in gore, and that readily has the ear of a public which is fascinated by the bizarre, the gruesome, the violent, the inhuman, the fearful. In 1987, Spiegel writer Wilhelm Bittorf described, possibly without really realizing it himself, this method of shock journalism:

AIDS has what the others are missing: nuclear death is anonymous, blind, impersonal, unimaginable even after Chernobyl, and thus dead boring. It may threaten to depopulate the earth, but that has little to do with the most private spheres of human experience. Even the worst environmental damage lies further away than the doom of infection in the erogenous zone. And if the Pershing rockets in [the German federal state] Baden-Wuerttemberg had only compromised the sex lives of the Germans, they would have been gone a long time ago.

Der Spiegel generated its own grotesque street ballads, like the story of the Munich German teacher, infected with AIDS through mere French kissing. I didn’t even have sex with him, the 2-year old said, bewildered. She cannot work anymore and is waiting for death. Or a woman from Dusseldorf, who purportedly destroyed her life during a holiday adventure in Portugal and lamented, I only slept with him once.

These stories clearly impede the search for truth, because they suggest that the conditions illustrated are true, although nobody has verified the facts in question-and much speaks for the fact that the illustrated conditions do not represent the truth.

And so, the simple and yet politically incorrect truth is rarely spoken out loud: the dreaded heterosexual epidemic never happened, Kevin Gray, of the US magazine Details reported to his readers in early 2004. The degree of epidemic in the population of developed nations has remained practically unchanged. In the USA, for example, since 1985, the number of those termed HN-infected has remained stable at one million people (which corresponds to a fraction of one percent of the population). But if HN were actually a new sexually transmitted virus, there should have been an exponential rise (and fall) in case numbers.
Additionally, in wealthy countries like the USA and Germany according to official statistics, poppers-consuming homosexuals have always made up around 50% of all AIDS patients, and intravenous drug users about 30%-a further seven percent are both. With this, almost all AIDS patients are men who lead a self-destructive lifestyle with toxic drugs, medications, etc. In contrast, the official statistics say that in poor countries:

- a much larger proportion of the population has AIDS
- men and women are equally affected and
- primarily, malnourished people suffer from AIDS

This clearly shows that AIDS symptoms are triggered by environmental factors like drugs, medications and insufficient nutrition. And it clearly speaks against the presumption that a virus is at work here that moves like a phenomenon of globalization-just like data streams, financial rivers, migration waves, jet planes fast, borderless, and incalculable, as the German weekly newspaper Die Zeit urgently warned on its front page in 2004.

Such a pathogen would inevitably have to attack all people in all countries of the world equally: men and women, straight and gay, African and German and not, as statistics reveal, in a racial and gender-biased way, attacking certain populations at different rates. In this context, Details writer Gray mentions a joke which made the rounds in the New York City Department of Health when the accumulation of AIDS statistics began: What do you call a man who [says he] got AIDS from his girlfriend? A liar!

In fact, the largest and best-conceived studies on the subject of sex and AIDS show that AIDS is not a sexually transmitted disease. The fact is glaringly obvious in the most comprehensive paper on this topic: Nancy Padians 1997 study on seroconversion rates among couples, published in the American Journal of Epidemiology with an observation period of ten years (1985 - 1995). In it, not a single case could be uncovered in which an HIV negative partner eventually became positive (or seroconverted) through sexual contact with his or her HIV positive partner. That is to say, the observed transmission rate was zero.

American virologist Robert Gallo and US Health Minister Margaret Heckler stepped in front of the cameras on 23 April 1984, with an important message: Today we add another miracle to the long honor roll of American medicine and science. Today’s discovery represents the triumph of science over a dreaded disease. Those who have disparaged this scientific search-those who have said we weren’t doing enough-have not understood how sound, solid, significant medical research proceeds.

The media immediately passed the news on to their audiences, without questioning what kind of medical research had led these scientists to believe what would soon become the dogma of the AIDS establishment: that AIDS can only occur in the presence of a viral infection, and that the virus dramatically destroys the patient’s helper cells (T cells). Gallo and Heckler then promised that an AIDS vaccine would be ready by 1986.

The public is still waiting for this promised vaccine. And the rest of us who have questioned the HN = AIDS theory are still asking for evidence of Gallos thesis that a virus is involved in the
onset of AIDS symptoms like the rare cancer Kaposi sarcoma, the lung disease PCP, herpes zoster, the deficiency-caused tuberculosis, and a growing number of other diseases and disorders added to the AIDS-related list yearly. Neither can the AIDS establishment explain why even AIDS patients in the end-stage have very few helper cells said to be infected with what is termed HN (although the orthodoxy precisely alleges that HN attacks and kills these T cells). For this reason, the collapse of the immune system cannot be plausibly explained by the HN = AIDS theory either. In 1985, the specialist publication Proceedings of the National Academy of Sciences drew attention to this helper T cell paradox.

Gallos papers were first printed in the journal Science weeks after the press conference. Thus, prior to his spectacular TV appearance, and for some days afterwards, nobody was able to review his work. This presented a severe breach of professional scientific etiquette, especially as review later showed that Gallos studies did not deliver any proof for the virus thesis.

But nobody opposed these very serious breaches of public trust. Instead, Gallo cast himself-surfing on the global wave of virus panic-as an infallible researcher. And the journalists believed him, so this virus-driven AIDS plan quickly embedded itself in the media, and from this time onwards it would drive all public information on AIDS. The words virus, cause, and AIDS were inseparably linked-and the world believed that AIDS is contagious. Scientific journalists around the globe were thrilled to have a great story about a sexually transmitted epidemic, not to mention a brave medical hero and savior in Robert Gallo.

The fact that most of the world fell for Gallos theory hook, line and sinker was confirmed in an investigation by Steven Epstein. The sociologist analyzed AIDS reports in leading specialist magazines in the opinion-shaping time from 1984 - 1986. It was shown that, among published texts referencing Gallos Science paper, the proportion that described the virus = AIDS hypothesis as a fact jumped from 3% to 62% between 1984 and 1986.

Expressions of doubt or skepticism [of the virus thesis]-let alone support for other hypotheses-were [in contrast] extraordinarily rare throughout this period from 1984 to 1986, Epstein argues. Findings such as these certainly support [culture critic Paula] Treichlers claim-that Gallo and his close associates established a network of citations that served to create the impression of greater certainty than Gallos own data warranted. In circular fashion, each article points to a different one as having provided the definite proof; the buck stops nowhere. This had a huge influence on the mass media (and with it on public opinion), which typically merely regurgitates information printed in Nature, Science or other specialist journals.

The reports of much of the mass media also influenced the content of scientific journals, according to a study published in 1992 in the New England Journal of Medicine. Even top scientists trust mass media sources like the New York Times, paper that often serves as the measure for other mass media. That is why editors often ask American journalists pitching their story ideas, Has the New York Times broken the story yet?

But, how objective and sound was the New York Times coverage of AIDS? Epstein also investigated this and found that in the specialist publications between 1984 and 1986, both the
proportion and the total number of articles in which it was blindly assumed HIV caused AIDS increased drastically.

The chief medical reporter for the New York Times, Lawrence Altman, distinguished himself as the leading media protagonist for the theory that AIDS is caused by HIV. Altman was so convinced of Gallos assertions that within weeks of the HeckJerGallo conference on 23 April 1984, he was using the neologisms AIDS virus and AIDS test even though Altman’s 15 May 1984 article acknowledges that, As the Red Cross and other studies progress, one of the most difficult questions that needs to be answered is: What does a positive blood test result mean? At this stage of AIDS research, scientists do not know if a positive test result means that the individual has an active infection, could transmit AIDS, had the infection at some unknown point in the past but recovered without becoming ill, or could still develop a fatal case at some future time.

Yet, no mainstream media reports have since answered this difficult question, and soon enough, it was simply dropped from public discourse. AIDS virus has become a synonym for HIV, just as AIDS test has replaced the more correct though still puzzling term antibody test even though Altman himself acknowledged some months later that scientists have not yet fulfilled Koch’s postulates for AIDS.

Both terms have firmly established themselves. This is highly problematical, however, because it allows scientific theories that have never been proven to pose as facts. In this case:

- That a virus called HIV causes the diseases grouped together under the term AIDS (Kaposi sarcoma, shingles, tuberculosis, etc.)

- That the existence of HIV antibodies can actually be proven with an HIV test Critics have questioned Altman’s objectivity and accused him of bias towards the Centers for Disease Control. In 1963, as a doctor, Altman joined the Epidemic Intelligence Service (EIS), which had been formed a few years after the Second World War. Altman was a high-ranking EIS scientist. And like the CDC, which is so fixated on the dangers of infections so that it has practically excluded other possible causes, such as chemical substances or toxins, the EIS has always been biased towards one goal: fight the viruses.

The EIS website information proudly claims that EIS pupils had discovered how the AIDS virus was transmitted. And so that as few people as possible leave the elite squad, its own alumni association fundamentally attempts to foster a spirit of loyalty to the EIS program through its activities.

The virus-fixated CDC, likewise, cannot be classified, in principle, as an objective information source at all. However, politicians and journalists continue to trust that any information the CDC makes public can be relied on without examination. For instance, in 2005, the German Süddeutsche Zeitung wrote: Worldwide, the Centers for Disease Control [CDC] in the USA are considered a model of a fast and consistently acting epidemic authority.

Altman, thanks to his high-level connections at the CDC, received various scoops from the
epidemic officials. And in 1992, he even openly admitted in Science that he had relied on the views of the CDC. And when the CDC was not confident to publish the story Altman didn’t think it was his papers [The New York Times] place to announce it. But strangely, nobody found it necessary to ask why the top medical reporter from the New York Times, who has a substantial influence upon the formation of public opinion, feels bound to follow the line of a federal authority.

In the mid-1980s, with fast-lane lifestyle theme cleared from the table to make room for the virus feast, there were no really weighty voices of opposition to the dominant views on AIDS. As social psychologist Elisabeth Noelle-Neumann fittingly argues, only members of a certain elite had the necessary influence upon people in power to decisively influence the formation of public opinion.

At the same time, excellence must appear early before the public eye, says Noelle-Neumann. And so it did, in the form of Peter Duesberg, member of the National Academy of Sciences, the USA’s highest scientific committee, and one of the best-known cancer researchers in the world. A critic of the first class had entered the ring to dispute the cause of AIDS. But Duesberg’s first major critique did not appear until 1987, in the journal Cancer Research—in other words, at a time when virus panic had already bombarded the public conscience for many years.

And, as those days and years ticked by, it became less and less likely that advocates of the AIDS virus theory would back-pedal, since they had already heavily invested, financially, personally and professionally, in HIV. Be it in the Spiegel, Die Zeit, The New York Times, Time or Newsweek—the AIDS orthodoxies theory had been championed everywhere. Researchers such as Gallo found themselves simply unable to retreat from their original claims because stakes are too high now, notes American journalist Celia Farber. Gallo stands to make a lot of money from patent rights on this virus. His entire reputation depends on the virus. If HIV is not the cause of AIDS, there’s nothing left for Gallo. If it’s not a retrovirus, Gallo would become irrelevant. And Gallo wouldn’t be the only one to sink into insignificance. Additionally, it would be very embarrassing to say that now, maybe, the antibody [test] wasn’t worth committing suicide for or burning houses for, states Farber.

And, in fact, numerous people, many of them completely healthy have killed themselves just because they tested HIV positive.

As with the polio epidemic, with AIDS the clear toxicological connections have been completely removed from the picture in the course of virus mania. Here, we must consider that there is no money to be earned with recreational drug-related hypotheses, which emphasizes poisoning by drugs, medicines and other chemical substances like pesticides. On the contrary, prohibiting certain chemical substances would cause huge profit losses for production and processing industries as well as the pharmaceutical, chemical, automotive and toy industries—and also for the media, whose existence is largely dependent on proceeds from these industry’s advertisements. In contrast, the virus theory clears the way for profits in the multibillions, with the sales of vaccines, PCR and antibody tests and antiviral medications. In the world of biomedical research, ties to industry are pervasive but mentioning the fact is not, writes William Booth in Science as
early as 1988. Correspondingly, new viruses are constantly invented—Ebola, SARS, avian flu, human papillomavirus (HPV)—to keep the cash flowing.

But doubts on the virus dogma were so clearly and comprehensibly formulated, that from the end of the 1980s, more and more people began to share in the criticism. Among them were several renowned scientists such as former Harvard microbiologist Charles Thomas, who founded the organization Rethinking AIDS at the beginning of the 1990s (renamed Reappraising AIDS in 1994 and renamed later again Rethinking AIDS). Thomas assembled hundreds of medical professionals, molecular biologists and other identified critics of the HIV = AIDS theory. Among them was Harvey Bialy, co-founder of the Nature offshoot Nature Biotechnology, and Yale mathematician Serge Lang (who died in 2005); like Duesberg, Lang was a member of the National Academy of Sciences (a list of more than 2000 critics is found on Rethinking-AIDS website, which re-formed in early.

It is good that the HIV hypothesis is being questioned, Nobel Prize winner for Chemistry, Walter Gilbert told the Oakland Tribune in 1989. Duesberg, Gilbert acknowledged, is absolutely correct in saying that no one has proven that AIDS is caused by the AIDS virus. And he is absolutely correct that the virus cultured in the laboratory may not be the cause of AIDS. There is no animal model for AIDS, and where there is no animal model, you cannot establish Koch's postulates. These arguments were so convincing, according to Gilbert, that he would not be surprised if there were another cause of AIDS and even that HIV is not involved.

Some time later, Gilbert expressed fundamental reservations in an English TV documentary critical of HIV/AIDS: The community as a whole doesn’t listen patiently to critics who adopt alternative viewpoints, although the great lesson of history is that knowledge develops through the conflict of viewpoints, that if you have simply a consensus view, it generally stultifies, it fails to see the problems of that consensus; and it depends on the existence of critics to break up that iceberg and to permit knowledge to develop.

The media prefer to make this consensus argument their own, even though it’s their duty to diligently research every medical claim, sort fact from theory and question even majority rule (however formed) to clarify every issue. But in 1990, for instance, even the venerable New York Times countered the provocative argument of alleged solitary dissenter Peter Duesberg when it claimed that virtually all of the leading scientists engaged in AIDS work believe that Duesberg is wrong. Yet, by 1990, as shown above, many renowned researchers said that mainstream research could not deliver any proof for their HIV = AIDS theory.

In 2000, Newsweek magazine expressed its incredulity that the consensus doesn’t impress the critics of the virus hypothesis in the article The HIV Disbelievers. Simultaneously, the piece calls the arguments of orthodox scientists clear-cut, exhaustive, and unambiguous. But evidence to support this statement could not be provided by Newsweek (not even upon request).

John Maddox, the editor at Nature from 1966 - 1996 led a personal campaign against critics of the HN = AIDS hypothesis. He even publicly censored Duesberg. On 7 November 1994 he justified this to the Spiegel, saying he found it irresponsible to say drug consumption is the cause of AIDS. Sir Maddox later contradicted this in a personal letter to Kiel internist Claus Kohnlein
on 20 September 1995, saying that he had not censored Duesberg because of his views but because of the manner in which he insists on expressing them. And Maddox added, that a hemophiliac relative of my wife died of AIDS.

But Maddox’s behavior—steering a scientific discussion in such a way based on personal views—is most frivolous and unethical. By doing this, he does no justice to his responsibility as Editor in Chief of Nature—a publication whose contents are taken at face value by the mass media.

Maddox took advantage of the huge influence of his Nature magazine again, at the beginning of 1995, when he published a paper by AIDS researcher David Ho, who claimed to have conclusively proven that HN alone causes AIDS. But critics ripped Hos paper to pieces. The quality of the data and the modeling were incomprehensible and about as convincing as a giraffe trying to sneak into a polar bears only picnic by wearing sunglasses, as Australian scientist Mark Craddock jokes in his detailed critique.

In turn, Nobel laureate Kary Mullis concludes: If Maddox seriously thinks or thought that these publications really prove that HN causes AIDS, then he should go outside and shoot himself—because if he had had no justification before, why did he reject all my possible explanations and alternative hypotheses? Why did Maddox have such a fixed opinion? Why did the whole world have such a fixed opinion? If it had taken until 1995 to find out what produces AIDS—how could everyone have known it for ten years? The facts are now on the table, and when one examines them closely, HN cannot be the cause of AIDS. There is no reason to believe that all these AIDS diseases have the same cause.

This staggering critique eventually found public validation in November 1996, when a paper was printed in Science that took the ground out from under the feet of Hos theses, according to journalists Kurt Langbein and Bert Ehgartner in their book The Medicine Cartel. The Science paper revealed that Ho had actually found no trace at all of the annihilating battle in the body between HN and the immune system, the connections of which the renowned scientist claimed to have discovered.

Unfortunately, few reporters in the mass media did the necessary homework before writing about HIV and AIDS. Instead, the papers were constantly packed with stories approved by the AIDS establishment, for which heroes and kings, traitors and villains are needed. And scientific journalists are particularly prone to striking up hymns of praise.

First came God, then came Gallo, decreed Flossie Wong-Staal, Gallos closest collaborator and consort in the Los Angeles Times in 1986. One year later, the Washington Post quoted Sam Broder, director of the American National Cancer Institute, as saying: Einstein, Freud—I'd put him [Gallo] on a list like that, I really would.

With David Ho, such excess was likewise not held back. On Christmas Day, 1996, just a few weeks after the journal Science had criticized the foundation of Hos work, the German Tageszeitung, without any irony intended, called him the redeemer and the long-awaited Messiah of the AIDS scene.
The reason for such jubilation? A catchy slogan with which Ho became famous in the mid-1990s, and which at least for a few years became the global chief doctrine for AIDS therapy: Hit HIV hard and early! It endorsed prescribing high dosages of antiretroviral medication as early as possible, even on patients testing HIV positive who do not show any disease symptoms.

A few days after his canonization by the Tageszeitung, Ho was celebrated on the cover of Time magazine as Man of the Year 1996. He was portrayed as a genius, whose brilliance had produced some of the boldest yet most cogent hypotheses in the epidemic campaign against HIV. [His] spirit is startling, manifested in a passionate transcendence [that] is evident in his gestures [Ho] is an extraordinary American success story. The Spiegel didn’t want to be out-of-step and soon declared Ho, thanks to his decided optimism to be the new shining light in the research world.

This euphoria did not last. In February 2001 even Altman had to admit in his New York Times that there had been an official turnaround in AIDS therapy and Hos concept (hit HIV hard and early) had to be abandoned. It had turned out that the medications were much too toxic, causing liver and kidney damage, and that their effects were immunosuppressive—in other words, they put patients Jives in danger.

Yet, even this defeat didn’t stop the Süddeutsche Zeitung from incorrectly writing at the beginning of 2004 that, Hos maxim hit HIV hard and early, with which he revolutionized HIV therapy, had led to patients having better chances of survival.

In 1987, the antiretroviral medication AZT became the first authorized AIDS medication. At the time, and for years afterwards, HIV I AIDS patients were typically given only one drug. This changed in 1995, when the multiple combination therapy (HAART) was introduced, in which, as is evident from the name, multiple substances are administered at the same time. Here, once again, the media broke out the streamers and confetti for another AIDS establishment party. For instance, Science declared the new weapons against AIDS as the breakthrough of 1996. And, it was universally reported that the antiretroviral preparations would help people with AIDS live longer, as the Washington Post announced in 2004.

Hans Halter from the Spiegel even gave concrete numbers: Those who are under the influence of medications, presently live on average 10 to 15 years. In contrast, the others who do not take any preparations only live five to ten years. These drugs generated billions of dollars in excess revenue for drug-makers: in 2000, global revenue was $4 billion; by 2004, it jumped to $6.6 billion, and in 2010, it should crack the $9 billion mark. For pharmaceutical giants, the preparations are bestsellers. At Roche, for example, Fuzeon, a medication that has been on the market since August 2004, triggered a 25% turnover increase.

But claims for the lifespan-increasing effectiveness of HAART medications are untenable. A close look at Halters comparison of survival rates, for instance, as gathered from the Arzteblatt (Medical Journal) for Schleswig-Holstein, shows that the average survival time for patients taking medication was four months in 1988 and 24 months in 1997. And according to CDC bulletins, it now amounts to 46 months -a long way from the 15 years mentioned by Halter. But
however big the increase in lifespan, one glaring omission is that everyone—doctors as well as patients—approaches the issue more carefully, because they have become ever more aware of drug toxicities.

Now, these drugs are often administered or taken with interruptions (so-called drug treatment holidays) and also in lower doses. The earliest example of this treatment about-face happened with the first AIDS medication, AZT, which, at the end of the 1980s, was still given in doses of 1,500 mg a day. But at the beginning of the 1990s, the daily dose was reduced to 500 mg, since even mainstream medicine couldn’t overlook the fact that the administration of higher doses led to much higher death rates.

Apart from that, we must soberly recognize that even a remaining lifetime of 46 months is not all that long, especially when you consider that perhaps millions of these medicated people are living with serious drug side effects that adversely affect quality of life. We must also recognize that there are these so-called long-term survivors or non-progressors. Common to these positive people is the fact that they have rejected AIDS medications from the start or only took them for a short time. Many of them tested positive more than two decades ago and are still living.

The AIDS establishment now calls these HN positive individuals who reject AIDS medications elite controllers as if they are somehow super-human. The establishment now claims that 2% of AIDS patients may fit this category, but only a large controlled global study (which is actually missing) would be able to determine the exact number of HN positive individuals remain healthy without taking AIDS drugs. However, the number of elite controllers is probably much higher, yet the vast majority of [so-called] HN-positives are long-term survivors! as Berkeley microbiologist Peter Duesberg states. Worldwide they number many, many millions.

A look at the CDC statistics before 1993 (and 2003 statistics from the Robert Koch Institute) shows that the number of AIDS deaths in the USA and also in Germany had already peaked in 1991, and decreased in the years following. And logically, the multiple combination therapy introduced in 1995/1996 cannot be responsible for this decrease. Newer CDC statistic, however, do show that the mortality peak lies approximately in 1995/1996. How can this be?

According to statistician Vladimir Koliadin, who analyzed the mortality data, this is due to the fact that in early 1993, AIDS in the USA was once again significantly redefined. From 1993 on, any individual testing HN positive with less than 200 CD4 cells per microliter of blood was counted as an AIDS patient. If both criteria were met, a diagnosis of AIDS defining diseases like shingles (herpes zoster) or Kaposis sarcoma was no longer necessary (although the old definition of, say, a positive HN test + Kaposis = AIDS was still valid).

This broadening of the AIDS definition meant that many people had the AIDS patient label superimposed upon them, even though they were actually not sick at all. A laboratory figure showing that an individual had less than 200 CD4 cells per microliter of blood was good enough for the AIDS establishment. But what this value ultimately means is, as discussed, anything but clear. Countries such as Canada have even decided not to introduce the CD4 cell count as criteria for the AIDS definition.
In any case, the number of AIDS cases in the USA doubled overnight as a result of the widening of the AIDS definition in 1993. This ensured the peak number of AIDS cases, and with it the mortality peak was pushed back (see diagram) from the early to the mid-1990s. If public and policy makers would have realized that epidemic of AIDS was declining, this might have resulted in reduction of budget for AIDS research and prevention programs, including the budget of the CDC themselves, according to Koliadin. Expansion of the definition of AIDS in 1993 helped to disguise the downward trend in epidemic of AIDS. It is reasonable to suppose that an essential motive behind the implementation of the new definition of AIDS just in 1993 was strong unwillingness of the CDC to reveal the declining trend of AIDS epidemic.

Even if we pushed all these considerations to the side, the introduction of combination therapy (HAART) and new active substances (particularly protease inhibitors) in 1995/1996 cannot explain the reduction in AIDS mortalities anyway; when the new substances were introduced, they were not available to even a good proportion of patients.

The opposite was probably the case. A meta-analysis with data from Europe, Australia and Canada shows that in 1995, patients used combination therapy during only 0.5% of treatment time. In 1996, the value lay at 4.7%, which is still extremely low. Former CDC director James Curran told CNN that, at the time, less than 10% of infected Americans had access to these new therapies, or were taking them.

Ten years later, while the media celebrated HAART's 10th birthday, the Lancet published a study that challenged the propaganda about HAART, showing that decreases in so-called viral load did not translate into a decrease in mortality for people taking these highly toxic AIDS drug combinations. The multi-center study—the largest and longest of its kind—tracked the effects of HAART on some 22,000 previously treatment naive HIV positives between 1995 and 2003 at 12 locations in Europe and the USA. The study’s results refute popular claims that the newer HAART meds extend life and improve health.

Commenting on the article, Felix de Fries of Study Group AIDS-Therapy in Zurich, Switzerland had this to say: The Lancet study shows that after a short period of time, HAART treatment led to increases in precisely those opportunistic diseases that define AIDS from fungal infections of the lungs, skin and intestines to various mycobacterial infections. De Fries also notes that HAART has led to no sustained increases in CD4 cell counts, no reduction in AIDS-defining illness and no decrease in mortality rates; its use is also associated with a list of serious adverse events such as cardiovascular disease, lipodystrophy, lactic acidosis, liver and kidney failure, osteoporosis, thyroid dysfunction, neuropathy, and cancers among users.

Yet, why even argue over pros and cons of HAART since statements about the life-prolonging effects of the medications are impossible to verify in the first place? Statements about the life-prolonging effects of the preparations are namely impossible, because the precedent condition has not been met: placebo-controlled studies. Since if one has no comparison with a group taking an ineffective preparation (placebo), it is not possible to know if the changes (improvement or worsening in patients health) are due to the medication or not. Placebo studies, however, have practically not been carried out anymore since the 1987 Fischl study published in. the NEJM, because, as it is said, the Fischl study found AZT to be effective.
For this reason, the AIDS establishment has since argued that it’s no longer ethically justifiable to withhold the (allegedly) lifesaving antiretroviral medication from the patients (not even in test series).

There are several objections, however, to this ethical argument. Not only do even leading orthodox AIDS scientist state that in medical science no researcher can assess a drug’s effectiveness with scientific certainty without testing it against a placebo. Also, as outlined, it was not HAART, but the huge widening of the definition of the disease as well as the drastic reductions in doses of AIDS drugs such as AZT that made the death rate from AIDS come down in the mid-1990s. Moreover, new studies show that most of the medical industry’s drug promises are false.

Pharmaceuticals hyped in glossy advertisements and TV commercials aren’t responsible for improving test patients health-rather, this can largely be traced back to the placebo effect. This is particularly worth noting when you consider that no expense is spared in bringing effective medications onto the market: expenditures for pharmaceuticals increased by 2,500% between 1972 and 2004—from $20 billion to $500 billion annually.

Moreover, two studies by the American Food and Drug Administration (FDA) make a case for the general introduction of placebo controls. This makes sense, since it is fully possible that proposed new drugs will have no effect at all. Or that, compared to the placebo, they are harmful; something that is also very possible, because medications are, as a rule, often connected with side effects—even fatal ones sometimes.

What right does the medical industry have to preach about ethics when its own human trials sweeps mortalities and physical damage under the carpet in the lust to get authorization to market their medications to the general public? In the USA alone, 3.7 million people—mostly poor Hispanic immigrants—have registered to participate in medical trials.

Lack of transparency and conflicts of interest continue to plague these drug trials, which are sponsored by the largest pharmaceutical companies in the world.

Even our most vulnerable citizens aren’t protected from the machinations of the medical industrial complex, as revealed in 2004. Infants as young as a few months old were experimented upon in US clinical trials, partly financed by pharmaceutical firms like GlaxoSmithKline, involving cocktails of up to seven medications. They were mostly black and Latino children from the poorest of circumstances gathered together under the auspices of institutions like the Incarnation Children’s Center (ICC) in New York; the ICC was even remunerated for supplying children for the tests. Stephen Nicholas, for example, was not only director of the ICC until 2002; he also simultaneously sat on the Pediatric Medical Advisory Panel, which was supposed to check the tests—which signifies a serious conflict of interest, criticizes Vera Sharav, president of the Alliance for Human Research Protection (AHRP), a medical industry watchdog organization.

These first-line Phase 1 and Phase 2 trials are associated with the highest health risk because they
are not meant to establish efficacy, so impact on the trial participants is highly unpredictable. These early trials aren’t meant to deliver an effective therapy, but rather, figure out how toxic the substance is (Phase 1) in order to then estimate if the active substance being tested has any effect at all (Phase 2). Biotechnologist Art Caplan explained that the odds are typically stacked against the drug: if Phase 1 trials prove that a substance is useful for an individual, this would have to be termed a miracle.

The children were suffering horribly from the side effects of the drugs tested on them, according to journalist Liarn Scheff, who broke the story in early 2004, on an alternative website. And children who didn’t want the substances were even forced to take them. For this, plastic tubes were sewn through the abdominal wall by surgeons, through which the substances can be directly injected into the stomach. The result: brain and bone marrow damage, blindness, strokes—and some children also died, according to Scheff. The New York Post seized upon the story and ran the headline: AIDS Tots Used as Guinea Pigs La term which the BBC also used for their television documentary Guinea Pig Kids.

In 2005, an official investigation ultimately came to the conclusion that government-funded researchers who tested AIDS drugs on foster children over the past two decades violated federal rules designed to protect vulnerable youths.

This finally prompted the New York Times, which is otherwise always the first on the scene on the subject of HIV/AIDS, to take up the highly explosive topic as well, with a decidedly different spin. In an article, two pediatricians were quoted as saying that, to have withheld promising drugs from sick children just because they were in foster care would have been inhumane, and there is impressive evidence that [the children] were helped [by the medications]. Details on this evidence, however, were never offered up. We even requested that authors of the Times article name the studies that prove these statements—but there was no response.

This might seem incredibly shocking, but it is all-too common in AIDS research. I have scoured the literature for evidence that the anti-HIV drugs actually prolong the lives, or at least improve the quality of the lives, of the children given these drugs—but I could not find any support for either possibility, says AIDS researcher David Rasnick. For example, the study Lamivudine in HIV-infected children by Lewis et al., not only has no control group but the authors also acknowledge that the [antiretroviral] study compound Lamivudine acts as a DNA chain terminator. And there is no data in the paper showing that the drug does anything good for the children. On the contrary, among the 90 children in the study, 11 children had to be withdrawn from the study for disease progression [in other words, it didn’t work for them] and 10 because of possible Lamivudine-related toxicity, and 6 had died.

But the AIDS orthodoxy continued along its own path, calling the clinical trials involving children so resounding in their success that the tests are now being spread out to Asia and Africa, according to Annie Bayne, spokesperson for the Columbia University Medical Center, which was also involved in the trials. This is not unusual, for AIDS research often goes into poor countries to carry out its medication trials. This is also true for trials of the efficacy of so-called microbicides, which are said to prevent the sexual transmission of HIV, and from which so much is promised.
Marvelous microbicides: [the] intravaginal vaginal gels could save millions of [human] lives, announced the Lancet in 2004, then qualifying their hopes by adding that, first someone has to prove that they work. Nothing has been proven at all, yet the miracle has already been announced far and wide. Experts, as the Lancet continues, were of the firm opinion that microbicides will only reach everyone who needs them if large pharmaceutical companies get involved. In the remotest part of Thailand you can buy a bottle of coke. We want microbes to be available like that.

This is all the more striking if you consider that the first microbicide tests of the active substance nonoxynol-9 (n-9) ended in catastrophe. At first, n-9 was also glorified by researchers as a microbicide with ideal potential microbicide because in vitro [test tube] studies pointed to its effectiveness. 900 sex-workers from Benin, the Ivory Coast, South Africa and Thailand were selected for a clinical trial, which involved smearing gel laced with n-9 into their vaginas. But the gel not only had no medical efficacy, as UNAIDS admitted, it also damaged the poor women’s epithelial cells.

If there is really doubt about whether a standard treatment is effective, the FDA should require that clinical trials of new treatments have three comparison groups new drug, old drug, and placebo, writes Marcia Angell, former Editor in Chief of the New England Journal of Medicine. For AIDS research, this meant that placebo groups had to be introduced to medication trials, for there were justified doubts that the efficacy of AZT (the standard AIDS treatment) had really been proven with the 1987 Fischl study.

Journalist and Harvard analyst John Lauritsen, who has viewed the FDA documents on the Fischl study, came to the conclusion that the study was fraud; the Swiss newspaper Weltwoche termed the experiment a gigantic botch-up and NBC News in New York branded the experiments, conducted across the US, as seriously flawed criticism which is not to be found in the rest of the mainstream media either because the statements of the AIDS establishment are completely trusted, or because, like the Neue Zurcher Zeitungs scientific editorial staff, one simply does not know of even such a significant study as that of Fischl et al.

The Fischl experiments were, in fact, stopped after only four months, after 19 trial subjects in the placebo group (those who did not receive AZT, but rather an inactive placebo) and only one participant from the so-called verum group (those who were officially taking AZT) had died. Through this, according to the AIDS establishment, the efficacy of AZT appeared to be proven. But the arguments don’t add up. A clinical trial observation period of only four months is much too short to be informative, considering the usual practice of administering AIDS medications over years, or even a lifetime and since long-term studies are missing in these and other medical research fields.

In the USA, for example, around $ 100 billion is spent annually on medical research. This figure has doubled since the mid-1990s, and almost a third of it comes from tax dollars. Yet long-term evaluations of pills and treatments are criminally neglected: just 1.6% of the $100 billion budget is allocated to long-term studies.
For patients taking medications, this is like Russian roulette, states British doctor Robert Califf.

The AZT study was financed by AZT manufacturer Welcome (today GlaxoSmithKline), which is clearly a conflict of interest. But somehow this, like the sloppiness of the Fischl study, didn’t bother anyone, especially not the pharmaceutical groups (nor the media!), for whom AZT would become a cash cow (it was actually said that AZT was worth its weight in gold).

Yet, the Fischl study’s double blind requirements (according to which, neither researchers nor patients were permitted to know who was taking AZT and who was taking the placebo) were violated after only a short time. In their desire to be given the alleged wonder-preparation, patients even had their pills analyzed to be sure that they were among the group receiving the medication and not the placebo; public propaganda had made test subjects believe that only AIDS medications like AZT could save them.

FDA documents also reveal that the study results were distorted, because the group that took AZT, and had to battle the adverse side effects, received more supportive medical services than the placebo subjects. For example, in the AZT group, 30 patients were kept alive through multiple blood transfusions until the end of the study-in the placebo group, on the other hand, this was only true in five cases.

There was widespread tampering with the rules of the [Fischl] trial-the rules have been violated coast to coast, said lead NBC reporter Perri Peltz in 1988, adding that if all patients with protocol violations were dropped, there wouldn’t be enough to be able to continue the study.

When preparing this report, we repeatedly tried to interview Dr. Anthony Fauci [probably the most powerful AIDS official in the USA] at the National Institutes of Health, reports Peltz. But both Dr. Fauci and Food and Drug Administration Commissioner Frank Young declined our request for interviews. These are the experiences of practically everyone who has criticized the theories of dominant AIDS medicine. The renowned British doctor and epidemiologist Gordon Stewart, for instance said: I have asked the health authorities, editors-in-chief and other experts concerned with HIV I AIDS repeatedly for proof of their theses-and I’ve been waiting for an answer since 1984.

Harvey Bialy, co-founder of Nature Biotechnology said: I am very tired of hearing AIDS establishment scientists tell me they are too busy saving lives to sit down and refute Peter Dues bergs arguments although each one assures me they could do it in a minute if they had to. We also contacted leading mainstream mass media and specialized journals including the New York Times, Time, Der Spiegel, Die Zeit, Stern, Tageszeitung, · Weltwoche, Neue Zürcher Zeitung, Nature, Science, Spektrum der Wissenschaft, asking them to send us clear evidence:
- That the existence of HIV has been proven
- That so-called HIV antibody tests and PCR viral load tests as well as the CD4 helper cell count specifically diagnose HIV I AIDS
- That HIV is the sole or primary cause of the diseases grouped together as AIDS
- That HIV is contagious and can be transmitted through sexual contact or blood
- That antiretroviral preparations are effective and prolong lifetime
- That the AIDS statistics proclaimed by the WHO and UNAIDS are sound
- That non-viral factors such as drugs, medications and malnutrition can be ruled out as primary causes.

But to date, not a single study has been revealed to us, not even from any of the many orthodox scientists and journalists we queried. This includes Nature writer Declan Butler, who wrote in the world-renowned journal in 2003: Most [mainstream] AIDS researchers strongly dispute these statements that there is no proof that HIV causes AIDS, that HIV is contagious, and so on. But Butler failed to respond to our request that he provide evidence of this in the form of relevant studies.

We also contacted John Moore of Cornell University in New York, who was quoted in Butlers Nature piece, and who thinks revisionists are best ignored. [They are leading] an unwinnable debate based on faith not fact But when we asked Moore if he could name the factual evidence for his HIV = AIDS = death-sentence theory, he responded by calling these critics the HIV-is-a-pussycat-fraction and charged them with pure stupidity and malice.

Scientific historian Horace Judson writes that, Central to the problem of misconduct is the response of institutions when charges erupt. Again and again the actions of senior scientists and administrators have been the very model of how not to respond. They have tried to smother the fire. Such flawed responses are altogether typical of misconduct cases.

These opinions were never known by the Fischl trial subjects. After four years, 80% of them had died; a short while later, all of them were dead. This is shocking but not really surprising, considering that AZT is an extremely poisonous chemotherapy-like medication, invented by researcher Jerome Horwitz in the 1960s. Hurwitz’s goal had been to develop a DNA blocker, which inhibits cell replication, to kill cancer cells. But, his test mice perished from the extreme toxicities of AZT.

On paper, [Hurwitz’s] logic was impeccable, [but] in reality, it simply didn’t work, summarizes Business Week journalist Bruce Nussbaum in his book, Good Intentions-How Big Business and the Medical Establishment are Corrupting the Fight against AIDS, Alzheimer’s, Cancer and More. When the experiment ended in failure, so, in a way, had the first half of Hurwitz’s life. Disgusted, he turned on AZT.

Horwitz himself said he was so cloyed with the drug that he dumped it on the junk pile. I didn’t [even] keep the notebooks. AZT was so worthless to him that he even didn’t think it was worth patenting.

AZT was in fact stored away instead of being dumped as toxic waste, and when AIDS mania surfaced in the 1980s, it was pulled out of the cupboard again. And the AIDS virus hypothesis, just like the many other virus theories for serious illnesses like leukemia, breast cancer and multiple sclerosis, would probably have disintegrated long ago, if not for AZT. In 1987, it became the AIDS therapy even though, in the recommended dosage, it was absolutely fatal. The medical community ignored the possibility that AZT-poisoning was the cause of death because they still had stuck in their minds the pictures of the first AIDS patients in the beginning of the
1980s, which certainly looked as if they’d been struck down and carried off by a deadly virus. So, when doctors looked at these AZT patients in 1987, they refused to make any connection with the highly toxic antiviral AZT. Their belief in the deadliness of HIV was so firm that they weren’t even shocked when all patients died within a short time. And so, with the Fischl study published in the NEJM, these doctors believed it worked and still allege to have tangible proof of AZTs efficacy.

HIV mania appears to cause its own range of symptoms: primarily a strong bias against the facts, including that chemical substances like drugs or prescription medications (particularly antiviral) are extremely toxic and can trigger precisely the observed symptoms (also mentioned on package labels) which they aim to prevent: destruction of mitochondria, anemia, bone marrow, and consequently immune system, damage, etc.

In the end, a vicious circle arises. Virologists have no proof of their thesis that a virus triggers the diseases grouped together under the term AIDS. So they consider proof to be collecting subjective information from clinicians who assert that the medications are effective. But, in industrialized countries, doctors very often treat patients not because they are sick (a large proportion have no physical complaints whatsoever), but rather because they have tested positive, they show only a certain number of helper cells or a slight so-called viral load has been measured via PCR. Virologists tell general practitioners that patients are carrying the deadly HIV.

The medications available for this, however, are highly toxic; their use produces an immune deficiency syndrome-and exactly fulfils the predictions of the virus hypothesis (that people will become severely ill and die). Healthy people are treated and worsening health is then attributed to the viral illness, which the drug therapy cannot counter.

Ultimately, if the medication doesn’t have any health-stimulating effects, this is also attributed to HIVs alleged craftiness; the virus itself is said to cause treatment resistant viral mutations. The patient dies with typical AIDS symptoms like dementia, wasting (weight loss), and neural damage. In their virus fixation, nobody imagines that the patient dies, not of AIDS, but of the very medical endeavors meant to heal.

Some HIV patients who are really sick do respond to antiretroviral medications. But this is because most of these patients suffer from what are called opportunistic infections (infections that occur as a result of an immunological/physical weakness, which in turn can have many non-viral causes). This means that they are infested by bacteria or fungi. In this context, antiretroviral treatment works like a shotgun therapy, destroying everything bound to DNA-including fungi, tubercle bacteria (Mycobacterium tuberculosis) and other microbes.

So, the therapy sometimes helps in the so-called AIDS end-stage. But it would actually be more sensible to treat opportunistic infections directly, with antibiotics and antifungal substances. The sensibility of such a treatment model was confirmed by a study published in the American Journal of Respiratory and Critical Care Medicine in 1998. HIV positive patients suffering from tuberculosis who received antiretroviral medications didn’t do as well as TB patients who received conventional treatment.
From an orthodox viewpoint, this is a paradox, so attempts are made to explain it away with the immune reconstitution theory. This explanation involves saying that patient’s helper cell counts rise (because HIV is purportedly repelled by antiviral preparations) but their physical condition worsens. At some point in the future, they postulate that patient’s conditions would then improve.

A look at the tables in the aforementioned studies, however, shows that increases in helper cells weren’t noticeable. Additionally, the health of many patients did not improve at all. On the contrary. And diminished health should be attributed to the damaging effects of the antiviral chemicals upon the immune system.

An effective tuberculostatic therapy (a combination of four antibiotics over six months, followed by a combination of three for three months) would then be preferable to an antiviral one. Instead, these days, patients are even administered both a tuberculostatic four-drug combo plus an anti-HN treatment: a chemical cocktail with toxic side effects that often enough cause death.

With conventional treatments, the medications are stopped after disease symptoms have subsided. But the belief in HN prevents this from happening with HN patients. At this point, the fixation on laboratory parameters comes into play again.

After an interruption in treatment, the viral load measured using the PCR goes up again. As shown, without any proof, mainstream AIDS doctors interpret this as a sign that HN has multiplied once again and re-attacked the helper cells with more force. So, antiretroviral medication is ordered once again. And when the patient’s condition worsens again, HN is blamed—and so the ultimately deadly preparations continue to be used.

Goethe knew that medicines could kill. Faust says: Here was the medicine, the patients died and nobody asked who convalesced. So we ravaged with hellish electuaries medicine worse than the pestilence in these valleys, these mountains. I myself administered the poison to thousands; they withered, I had to witness that the brazen murderers were praised.

Even celebrities fall for the theory that antiretroviral substances like AZT are the only hope in the battle against AIDS. Take, for example, Freddie Mercury, former front man of British rock band Queen, who was bisexual and had himself tested during the general AIDS panic at the end of the 1980s. The result: positive. Mercury was terrified and took his doctors advice to begin taking AZT. Mercury belonged to the first generation of patients, who received the full AZT load (1500 mg a day). At the end, he looked like a bone rack, and he died in London on 24 November 1991 at the age of 45.

The Russian Rudolph Nureyev, held by many to be the greatest ballet dancer of all time, also began taking AZT at the end of the 1980s. Nureyev was HN positive, but otherwise he was completely healthy. His personal physician, Michel Canesi, recognized the deadly effects of AZT and even warned him about the drug. But Nureyev proclaimed, I want that drug! Ultimately, he died in Paris in 1993 the same year that former Wimbledon champion Arthur
Ashe met his maker at the age of 36, after he had been declared HIV positive in 1988 and his doctor prescribed for him an extremely high AZT dose.

At some point, Ashe discussed AZT's toxicity. In October 1992, he wrote a column for the Washington Post. The confusion for AIDS patients like me is that there is a growing school of thought that HIV may not be the sole cause of AIDS, and that standard treatments such as AZT actually make matters worse, Ashe acknowledged, adding, there may very well be unknown co-factors, but that the medical establishment is too rigid to change the direction of basic research and/or clinical trials. Ashe wanted to stop taking AZT, but he didn’t dare: What will I tell my doctors? he asked the New York Daily News.

What Ashe didn’t have the heart to do-resist the pressure of prevailing AIDS medicine and decide against AZT intake-apparently saved the life of basketball megastar Earvin Magic Johnson.

At the end of 1991, Magic shocked the world with the news he had tested HIV positive. It can happen to anybody, even Magic Johnson, said Time magazine on 18 November 1991. A few days later, Time wrote that the basketball player had put the risk of heterosexual transmission squarely in center court. But what was the basis of this assumption? Nothing at all, for the American magazine-just like the rest of the media world-simply referred to Johnson’s mere conjecture that he had picked up the AIDS virus heterosexually, that is to say through sex with a woman.

Evidence to support this statement is not available. Magic Johnson had tested positive, but at the same time, he was the picture of health-until AIDS ruler Anthony Fauci and his personal doctor, the New York AIDS researcher David Ho, insistently advised him to take AZT. Johnson followed their advice.

But Magic’s health rapidly deteriorated, so much, in fact, that he felt like vomiting almost every day, according to a 1991 National Enquirer story Magic Reeling as Worst Nightmare Comes True-He’s Getting Sicker. But virus mania was by then so dominant that nobody thought that the extremely toxic medications could have caused Magic’s serious health problems.

There was not a lot of time to think about it anyway, as Johnsons symptoms suddenly disappeared after a short time. In the summer of 1992, after the media announced his retirement from basketball in late 1992, he even led the US basketball team to the gold medal at the Olympic games in Barcelona. This was a grandiose achievement, and had he still been under the influence of AZT, there was no way he could have accomplished such a thing.

One assumes, then, that Magic only took AZT for a very short time; when he discontinued the medication with the deadly side effects, his complaints likewise disappeared. Indeed, years later, in 1995, he admitted in a personal conversation in Florida that he had only taken AZT for a very short time. The medications were connected with far too severe side effects. And so came the saying, There is no magic in AZT, and no AZT in Magic.

But AIDS drug manufacturers also play a highly competitive game in an increasingly marketing-
driven industry. For several years GlaxoSmithKline (GSK) used Magic Johnson to spread its miracle cure messages especially among urban blacks. The basketball stars image is splashed on billboards, subway posters and full-page ads in newspapers and magazines. The ads picture a robust-looking Johnson and feature messages such as, Staying healthy is about a few basic things: A positive attitude, partnering with my doctor, taking my medicine every day. Those ads are now gone because Johnson got a better offer from Abbott and is now promoting another combination AIDS drug, Kaletra.

However, this does not necessarily mean that Johnson himself is taking these highly toxic drugs. As outlined, the opposite is obviously true. Magic is the poster boy for HIV positive heterosexuals and he’s a spokesman for a drug manufacturer, so he has a financial conflict of interest that may disallow him from revealing if he is really taking GSKs Combivir or Abbotts Kaletra and, if so, how much drug he’s really taking. Johnson has not directly confirmed that he is taking the drugs he pushes, says AIDS drugs researcher David Rasnick.

In October 2004, we approached the Magic Johnson Foundation to ask if the basketball player has taken any AIDS medications since the Olympic triumph in 1992, and, if so, for how long. But, as of today, we have not received a response.

The publication of the Darby study in September 1995 in Nature also contributed to the cementing of the belief that AIDS is a viral disease. In it, death rates of hemophiliacs in England who had tested HIV positive were compared with those of their HIV negative hemophiliac counterparts over a period from 1985 - 1992. The printed graph showed that the death rate of positive-tested hemophiliacs began to rise from about 1986; in 1987 it rose even more sharply. In comparison, the graph showing HN negative hemophiliacs remained practically unchanged. Orthodox medicine claimed that this was proof that these deaths were caused by HN.

But this study stirred up sharp criticism. Previously mentioned Australian researcher Mark Craddock, for example, penned a decisive paper and submitted it to Nature. But it was rejected—along with papers by Peter Duesberg and the Australian Perth Group—even though the logic behind their critiques is impressive.

Hemophiliacs lack coagulation factor VIII and a replacement has been available since the 1960s causing hemophiliacs life expectancy to continuously rise until 1985, right when HN antibody tests were introduced. This is a decisive factor, negligently missing from the Darby study. The HN antibody tests introduced in 1985 were immediately and massively deployed. At the same time, the whole world memorized the formula: positive test= HN infection = AIDS = death sentence. Because of this, the rise in hemophilic’s death rates is easily explainable. Those who received a positive test result were put into a state of shock and many committed suicide. The rest, regardless of their health status, were automatically treated as AIDS patients. Researchers and doctors tried out all sorts of toxic substances on them, administering them long-term, including antifungal medications or Eusaprim, an antibiotic that hinders cell division. This also affected hemophiliacs who had tested positive but otherwise didn’t have any health problems—until they started taking the toxic AIDS medications.

We can’t be sure exactly which medications were administered to those declared AIDS patients, since they weren’t listed in detail, as Nature editor John Maddox confirmed in 1995. But, the
Spiegel reported in 1985 that, more than a dozen different medications are in clinical trials in the United States alone—all of them have shown little success so far, and are burdened with severe side effects. Even HPA 23, the substance favored by French scientists and developed at the Louis Pasteur Institute, and with which Rock Hudson was treated last autumn, has its difficulties. In Paris, a clinical study of HPA 23 is being carried out on 33 subjects; but, the medication had to be discontinued with numerous patients because of extreme blood and liver damage. In 1987, AZT busted onto the market and all positive patients, including hemophiliacs, immediately received the medication associated with fatal side effects—something that explains why hemophiliac’s death rates sharply increased from this point onward.

Incidentally, Rock Hudson died in 1985, officially of AIDS. Less well-known is the fact that Hudson’s male partner had tested negative and had no AIDS symptoms something which clearly speaks against AIDS being a viral disease. In the mid-1990s, American congressman Gil Gutknecht became aware of this and all the other inconsistencies and shortcomings of the HIV = AIDS hypothesis. And so he confronted the AIDS establishment’s highest operatives with a whole range of critical questions, including: Where is the proof that clearly shows that AIDS is a contagious disease?

But Gutknecht never got a real answer either. Incidentally, the blood plasma designed for hemophiliacs is freeze-dried before its administration, often for long periods. If you hypothetically assume that this virus does exist, it would not survive such extreme conditions, as mainstream medicine admits. The Centers for Disease Control states that this drying process of human blood or other body fluids reduces the theoretical risk of environmental transmission to that which has been observed—essentially zero. Incorrect interpretation of conclusions drawn from laboratory studies have unnecessarily alarmed some people.

No surprise, then, that in specialist literature, there is not one single clear-cut case of HIV infection among health care workers who typically deal with blood on a daily basis. As statistics on HIV infection remain stable or decrease in developed nations, the AIDS establishment and the media turn their focus to Africa. Headlines and TV news stories are scary: millions of Africans have died and will die from HIV I AIDS. But in reality, these are computer-generated estimates from the World Health Organization (WHO), based on a highly questionable data pool. And they seem grotesquely exaggerated when one compares them with the population statistics of precisely those countries where depopulation has been predicted for many, many years.

Botswana has just concluded a census that shows population growing at about 2.7 per cent a year, in spite of what is usually described as the worst AIDS problem on the planet, writes South African author Rian Malan in a cover story for the British news magazine The Spectator: Africa Isn’t Dying of AIDS. Malan points out that there is similar bad news for the doomsayers in Tanzania’s new census, which shows population growing at 2.9 per cent a year. Professional pessimists will be particularly discomforted by developments in the swamplands west of Lake Victoria, where HIV first emerged, and where the depopulated villages of popular mythology are supposedly located. Here, in the district of Kagera, population grew at 2.7 per cent a year before 1988, only to accelerate to 3.1 per cent even as the AIDS epidemic was supposedly peaking. Uganda’s latest census tells a broadly similar story, as does South Africa’s.
AIDS is a huge business, possibly the biggest in Africa, says James Shikwati, founder of Inter Region Economic Network, a society for economic promotion in Nairobi (Kenya). In a 2005 interview with Spiegel editor Thilo Thielke, Shikwati added that, nothing else gets people to fork out money like shocking AIDS figures. AIDS is a political disease here: we should be very skeptical. But the people in the control centers of politics, science and media aren’t suspicious, so they ignore the extreme discrepancy evident between perpetual predictions of horror (Africa will be depopulated by AIDS) and actual population increases.

It is still firmly assumed that the HIV antibody tests, which are an important basis for the WHO’s AIDS projections, are reliable measurement instruments. But let’s take a closer look back to 1994. At that time, the Journal of Infectious Diseases published a paper on HIV tests with lepers in Zaire, compiled by no less than Max Essex, who is said to be one of the founding fathers of orthodox AIDS science, and of the theory that HIV or AIDS originally comes from Africa. Essex observed that lepers reacted positively to the HIV test. For this reason, Essex points out that the results of the tests should be taken with a grain of salt—above all for patients suffering from diseases like leprosy or tuberculosis. And in places where these diseases are so widespread, particularly in central African cities, antibody tests are probably insufficient to define an HIV infection without any doubt. Essex thought it best to let this observation count for all African countries.

Neville Hodgkinson, then medical correspondent for the Sunday Times jumped on the topic and spent weeks traveling through Africa. When I asked people what disease they were dying of, they replied: from AIDS. Whereupon I inquired: but from which disease in particular? To this they said: This patient has tuberculosis, that one chronic diarrhea, this one malaria and that one leprosy—all diseases that have been known in Africa for ages. But then everything was re-diagnosed as AIDS out of fear of AIDS.

Nobel laureate Kary Mullis adds that, They got some big numbers for HIV positive people [in Africa] before they realized that antibodies to malaria—which everyone in Africa has—show up as HIV positive on tests. And not only malaria, but also dozens of other typical illnesses like chronic fever, weight loss, diarrhea and tuberculosis cause positive test results.

The HIV/AIDS epidemic is actually a smorgasbord of well-known diseases, many of which correlate closely with poverty. You can’t speak concisely about AIDS in Africa without featuring the subject of poverty. Yet, this is still criminally neglected in a region where a third of the population is malnourished and more than 30% of babies are born underweight. As we know, malnutrition has devastating effects upon health, and is a decisive factor in many diseases such as tuberculosis.

At least The Lancet took on this topic in 2004 and printed an article titled: Preventing HIV/AIDS Through Poverty Reduction. This document praises South African president Thabo Mbeki (who is generally sharply scolded for his critical position towards the AIDS establishment) by pointing out that Mbeki has highlighted poverty as a factor contributing to the spread of the epidemic, [and] it is useful to consider the role of poverty as a factor contributing to it, and the implications of this for prevention efforts.
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 Hoofnagles 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.

This is no surprise, since substances like interferon ultimately work like chemotherapy and for that reason can severely affect more than just the liver; 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.

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. 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. 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.

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 bloodstream 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.

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. 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. 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 HN, 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? 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.

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. 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.

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).

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). 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 (IDUs) who had no access to the programs. The researchers concluded that these programs do not help to prevent a so-called HCV infection. 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 its 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!), 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. 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 medications life-prolonging effects are impossible.

Just as with HIV I 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 viruses-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.

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. 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.

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.

Another worthy investigation would be to look as 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. Its 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 panicles 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. 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.

But all these studies are irrelevant because they prove that the medications do not heal and, even worse, that they cause harm. A few years ago, a large American study was published in the Annals of Internal Medicine. 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) 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.

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. Rutland’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.
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 diseases 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. Anderson disclosed that she believed she had been infected by her ex-husband, drummer Tommy Lee, when they were tattooing each other.

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. 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
The hysteria caused by the alleged bovine epidemic BSE (Bovine Spongiform Encephalopathy which is a sponge like 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.

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 of the 1990s, there was still talk of a time bomb effect and the death of up to ten million people was still held as a possibility. But in 2001, the British Medical Journal called it Creutzfeldt-Jakob disease: the epidemic that never was and at the beginning of 2005, a British research team gave the all-clear and reported: Creutzfeldt-Jakob Disease Is Cancelled.

In reality, a giant BSE bureaucracy was erected, which registers every twitch in the stable and tests every one of the butchers slices, according to the Frankfurter Allgemeine Sonntagszeitung. The program came with a hefty economic price; BSE hysteria has cost Germany at least € 1.5 billion to date, said Sucharit Bhakdi, Director of the Institute of Microbiology and Hygiene at the University of Mainz (his comments appeared in 2002, it is worth noting): And yet, the obligatory BSE tests on cattle were completely pointless and a pure waste of money. Among the 5.1 million tested cattle, just 200 sick animals were found. And these 200 BSE cattle could have infected three people at most, and that over the next 30 years, states Bhakdi. His
advice: do nothing. It is completely sufficient to do just that when (so-called) infected animals are taken away.

Since then, virus mania has continued to plague the beef industry. Companies like the Swiss firm Prionics, which controls 50% of the world market for BSE tests/continue to make millions (ultimately at a cost to the consumer). The belief that an infectious particle, or more precisely a prion (proteinaceous infectious protein) makes cattle sick is still firmly anchored in the public conscience. And yet, since the beginning of the 1990s, data has been diligently collected and published—but despite all efforts, there is still no real proof of the hypothesis that a deformed protein (prion) has infectious properties and is capable of causing brain-softening (spongiform encephalopathy): BSE in cattle, and the new variant Creutzfeldt-Jakob disease (vCJD) in humans.

The atomic structure of these allegedly infectious prion proteins isn’t even known. BSE is termed an epidemic, but this is wrong—just as the presumption that BSE is contagious is also wrong, writes Anton Mayr, Chair of Microbiology and Epidemiology at the University of Munich. And even BSE transmissibility to humans, neither with classical Creutzfeldt-Jakob disease (CJD for short) nor the new current form, new variant CJD or nvCJD, has not been proven.

Depending on the spirit of the times and which authorities are in power, one dogma or another dominates the scientific scene, often with an exclusivity that does not admit any other possibilities and hinders new ideas, writes Roland Scholz, BSE: The Epidemic That Never Was Professor of Biochemistry and Cellular Biology in Munich, and a critic of the dominant BSE theory. And in the BSE drama, this dogma is infection. Here, Nobel Prizes can play a controlling and unhealthy role. On the one hand, these awards usually follows the spirit of the times, i.e. along conventional lines of thought. On the other, they can cement paradigms.

Into the 1960s, scientists were of the opinion that encephalopathy in sheep (known as Scrapie, because the animals constantly scratch themselves) only occurred endemically, that is, only within certain flocks. In which case, up to 30% of a herd can be afflicted. Scrapie [sheep disease] is said to be a genetic disease that can be eliminated by establishing adequate breeding protocols, according to research done by Herbert Parry in 1962.

But after the awarding of the Nobel Prize in 1976 to the previously mentioned researcher Carleton Gajdusek Scrapie, like all spongiform encephalopathies (softening of the brain), was redefined as an infectious disease. It was reclassified after Gajduseks 1970s research on dementia observed in the population of Papua New Guinea; he declared this sponge-like brain disease (spongiform encephalopathy; BSE is also classified as one) to be a viral disease transmitted through food.

The sneaky virus culprit, however, could not be found. Nonetheless, microbe obsessed research continued to hold tight to its pathogen theory. Virus hunters were desperate to impose the contagion theory onto dementia as well.

The work of Stanley Prusiner served as a basis for this theory. In 1982, he succeeded in
identifying plaques (accumulations) in the brain, which are characteristic of a brain suffering from neural damage—and which are said to be the cause. In these plaques, certain proteins called prions are found, which primarily build up on neurons, in an abnormally altered structure (the P-pleated sheet structure). Whereas, the normal (healthy native) prion protein shows predominantly spiral-shaped α-helix structures and hardly any abnormal p-pleated sheet structures.

The speculative plaque development model implies, then, that prion proteins with an abnormally altered P-pleated sheet structure are the source of plaque formation. The idea is that, as particles foreign to the body, they succeed in getting into the host. Upon arrival, they impose their deformed P-pleated sheet structure upon the normal protein with its α-helix form. And this P-structure makes it easier for prion proteins to clump together, so plaques accumulate on the neurons and jam neural receptors. These plaques can then only be degraded with difficulty. This process gradually leads to a build-up of molecular waste in the brain, causing the death of increasing numbers of neurons. The holes that develop through this, as well as the deposits between cells (vacuoles), give the brain the sponge like appearance so typical of the disease (the term spongiform encephalopathy comes from the Latin spongia = sponge).

In 1987, Prusiner succumbed to temptation and brought his till then largely ignored prions into the epidemic game, something that brought him an enormous degree of recognition. Ten years later, in 1997, he was even ennobled with the Nobel Prize, as the Deutsche Artzteblatt wrote. With this, the infection topic had been cemented. The Prusiner prion was declared to be the trigger for spongiform brain diseases, and was said to be more dangerous than all previous infectious agents.

So dangerous that it is allegedly impossible to deactivate it by the usual means (heat, radiation, chemical substances). For with the prion, a protein was branded as infectious evil-doer for the first time; it is said to be especially dangerous because the immune system can’t fight it off, since it occurs naturally in the body and is not a foreign substance. Note that, according to this theory, plaque formation is initiated by abnormally structured prion proteins from a foreign organism; these then clump together with healthy prion proteins in the new organism to form plaques; these plaques and the prions found in them are composed of proteins occurring naturally in the body.

In 1986, as the BSE epidemic hysteria began in Great Britain, health authorities believed in an infection involving a pathogen transmitted through feed. Without having any detailed evidence at hand they speculated that prions were present in the sheep suffering from brain-softening (Scrapie). These prions were said to have subsequently managed to reach cattle by way of the meat and bone meal (which contained waste from slaughtered sheep) used as cattle feed.

Through this, it was said, the cattle became sick. And so a mere conjecture quickly became a hypothesis that was blown up into a threatening scenario in the interplay between the media and certain scientific circles.

The media plays a fatal role, because, in its tendency to come to short-term sensationalistic clear statements, it often feigns a clarity—or a threat, that really is not supported by scientific findings, says Jürgen Kronig, England correspondent for German weekly newspaper Die Zeit, in criticism of his own profession. The media had decisively contributed to hysterical public reactions, which
in turn brought the political and scientific establishment to hasty action. Pictures of stumbling cattle and of cow carcasses being shoved into incinerators further fueled the flames of hysteria. Prions became the horsemen of the apocalypse that threaten humanity.

The illustration describes the model of the alleged infectiousness of the prion protein. If the protein aggregates that have developed in a spongiform-altered brain are injected directly into a healthy brain, they trigger an accelerated aggregation process in similar proteins in this brain. Through protein-protein interaction, the aggregate causes membrane protein molecules to be rearranged from the healthy or normal helical into the β-pleated sheet form, and allows them to accumulate on the aggregate, which gradually grows to the size of a plaque. Prusiner first called this amplification, but not long later he (false) renamed it infection, because it sounded dangerous.

The scientific community just parrots his theory without analyzing how the infection arises, or whether a simple immune reaction against foreign proteins might not possibly have left its histological traces (as researcher Alan Ebringer claims, this phenomenon has been known as EAE for decades). Apart from that, the aggregate shown in this diagram, which is said to have entered the brain as an infectious agent, did not enter the body orally (not through food), but rather through an intracerebral injection (directly into the brain). And this is of course not the way that animals in the wild or humans become infected.

Incidentally, in the dim and distant past, Prusiner introduced c and Sc before he clouded up terminology with his prion or prion protein. c stands for cellular, and for the normal membrane protein, which occurs in α-helix form (more precisely: whose neutral position is the helical form), and which is now thought to be an extracellular superoxide dismutase, which protects cells from oxygen radicals produced extracellularly (outside the cells). Prusiner gave this membrane protein the name PrP (prion protein), and he called the resulting infectious agent prion. Sc stands for Scrapie: the membrane protein which is found as an aggregate in Scrapie sheep, the primary structure (amino acid sequence) of which is identical to that of normal membrane proteins (to c), but which has a different secondary structure (pleated sheet instead of helix) and could accumulate for this reason.

According to Prusiner's conception, the aggregate of Sc first forces the normal helix-shaped c into the pleated sheet form. But anyone who knows a bit about proteins knows that a native protein does not have an absolutely stable structure, but rather fluctuates between various states: with the membrane protein in question, there’s a constant fluctuation between c and Sc. Whether an aggregate actually forces the normal c-proteins to transform into Sc and then to clump together with the aggregate (in other words, whether it functions like a catalyst that initiates a process), is a hypothesis—or better, pure speculation.

But with a little critical analysis, we see the deep rift between truth and illusions. The food industry has conveyed to the public an incredibly distorted picture of food production since the 19th century, through advertisements and public relations. Truth matters little in this spin doctoring, and is massively impeded by the attempts of all sorts of cliques and interest groups to get maximum profit.
I think that primarily to blame [in the BSE disaster] are the agricultural ministers, who have a sort of symbiotic relationship to agro-business: to the large corporations, not just the meat feed manufacturers, but the chemical groups as well, says Kronig. Through this, research was contaminated from the onset: this means the experts were directed too much by their interests. The research was not carried out openly. This has to change, for only when there is absolute clarity over the reasons, can something sensible really be undertaken.

How tightly research and big business are interwoven can also be seen in the example of Nobel Prize-winner Prusiner, who has developed his own BSE quick test and promoted it far and wide through an article published in the scientific journal Spektrum der Wissenschaft in early 2005. Prusiner did not hesitate to emphasize that the test could possibly also be suitable for testing human blood for SSE-something that, if it became reality, would mean that the test manufacturers had the equivalent of a money tree in their hands. One can only agree with Prusiner when he himself writes in his article: One may suspect that I propagate the thorough CDI test [Prusiner’s quick test] in my own interests.

So the theory goes that prions have spread across the borders of species (for example from sheep to cow). And researchers concluded that if prions can manage the jump from sheep to cow, then humans could also become infected from beef products.

But there are numerous flaws in the experiments upon which these hypotheses are based. Extracts from the brains of animals with neural diseases were directly injected into the brains of test animals. When, after a year, they detected the existence of the nerve-damaging accumulations (plaques) and holes in the brains, it was taken as proof that a prion had caused an infection, which in turn had caused the development of the plaque.

But the alterations in the brain could also have another cause. They could be consequences of an immune reaction, for instance, with which the body defends itself against foreign proteins (in this case the foreign prion proteins). However, researchers didn’t consider this at all, even though a 1998 study by immunologist Alan Ebringer of Kings College, London pointed out the possibility that many experiments involving injecting brain material from animals suffering from encephalopathies into the brains of healthy animals didn’t necessarily cause the transmission of Scrapie or BSE (as is held to be the case); even if these animals did later develop neurological symptoms and plaques were found in their brains.

We must also remember that laboratory experiments in which cerebral matter is directly transmitted from one brain to another proves nothing in terms of infection, since this is supposed to occur via the mouth (orally). When was the last time your brain came into contact with someone else’s brain mass?

Ebringer: The Prion-research workers do something that is not allowed. They inject brain tissue homogenates into experimental animals, and when neurological symptoms appear they say they have transmitted BSE. However, they have done nothing of the sort, because what they are doing is producing experimental allergic encephalomyelitis (EAE). I think all prion experiments involve production of EAE and not transmission of BSE.
An additional mind-boggier is that the prion experiments involved no proper control experiments (involving a comparative group of animals that are injected with something that can be compared to what the original test subjects receive). In 2004, a paper was published in Science claiming to have produced a sort of irrefutable proof for the prion infection = brain-softening theory. In the experiment, brain extracts from infected animals were not injected directly into the brains of the test mice. Instead, a deformed prion with a -pleated structure was artificially produced, and it was assumed that this structure would give the prion an infectious property. Then this prion protein with the -pleated structure was injected into mouse brains. After one to two years, the mice developed neurological disorders.

But, once again, the experiments have no scientific value. Not only because neurophysiology and immunology differ between mice and humans, so results can be fundamentally misleading. Also, as with many experiments conducted by the guild of prion researchers, there were no control experiments involving an extract that can be compared to the originally administered fluid. The salt solution alone, which was injected into the brains of the control animals, is not a true control. The researchers should have taken at least one other solution containing a protein and have introduced it into the brains of the test mice. Or, even better, a genetically engineered prion protein that did not have the -pleated structure, but rather the healthy/normal a-helix form. Defendants of the prions in meat and bone meal hypothesis also refer to tests in which raw brain material is fed to laboratory animals. But raw brain that comes from brain-diseased animals cannot be equated with animal feed meal, since these substances have completely different contents. Here as well, the test results cannot be carried over to reality. Furthermore, adequate control groups are missing from these experiments as well (groups of animals that are fed healthy cow brain).

For this reason, it cannot be asserted that a certain constituent in the brain material fed to the mice (a deformed prion, for example), had produced alterations in their brains after a year or more—or if the brain material itself had not been responsible. For this reason, the observed symptoms can also be interpreted as portraying the results of an immune reaction.

Of course, experimental games and speculation are perfectly suitable for impressing gullible research colleagues, politicians, journalists and the public. But, they are scientifically worthless. For no controlled feeding experiments in the field exist studies that anyone with a healthy dose of common sense would require, and which everyone believes have long been carried out by inventors of the meat and bone meal hypothesis, criticizes Roland Scholz.

This means, a large herd should have been separated into two halves: one group receives meat and bone meal and the other doesn’t receive this feed. Since this has been neglected, however, the conclusion is evident: it has not yet been shown that cattle become infected with BSE by being fed meat and bone meal. That an infectious protein in meat and bone meal triggers BSE is still an unproven conjecture. Incidentally, it would have been even more informative, if a controlled experiment had been carried out with specifically manufactured meat and bone meals (consisting of material from Scrapie sheep or BSE cattle), something that, incidentally, could still be done. Then one could figure out whether the meat and bone meal is a trigger at all—and if
so, what kind of infectious agent it was-or if a change in the animal meals manufacturing process could possibly have been the cause.

Due to the lack of proof for the thesis that prions in meat and bone meal can trigger the bovine disease BSE, it seems particularly advisable to keep an eye out for other attempts at explanation as well. It could very well be that a defect in the genetic make-up of cattle from a few British herds was multiplied to such an extent through over breeding that the animals became ill.

BSE manifests primarily in young cattle aged two to five years (cattle can live up to 25 years), while most diseases comparable to BSE tend to appear at an advanced age. With the rare disease called mad cow disease, the animals were considerably older. And with humans as well, these spongiform encephalopathies (brain softening) that do not appear within families are typically age-related diseases. But children and adolescents also come down with the spongiform encephalopathies, which can be frequently observed within families.

With modern high-performance cattle breeding, most cows are descended from only a few bulls that are often related to each other. Thanks to artificial insemination, the semen of a single bull is said to guarantee high-performance cows as daughters and can supply an entire region. Incest should be avoided, but with breeding geared only towards high performance-in England, a cow provides 60 - 70 liters of milk daily-this rule is usually not observed. A single bull in a region's insemination institute could then be the father of many of a district's cattle herds, and simultaneously also their grandfather, writes Roland Scholz. With this, what has been usual in flocks of sheep for centuries has arrived in cattle herds over the past few decades.

With spongiform encephalopathies, the paradigm shift from infection to genetics could have been executed with Prusiner. In his investigations into the cause of SE on a molecular level, he found that a certain membrane protein on neurons (prion) had a tendency to reshape from the functional/sound a-helix form into the functionless 13-pleated sheet form.

These 13-pleated sheet proteins shaped like corrugated metal tend to clump together with other proteins that likewise feature a 13-pleated sheet structure. The aggregates grow, develop the plaques (clumps) on the nerve cells typical of brain softening, and can then force other prion proteins to re-shape: first on the same cell, then on neighboring cells, so that the process spreads throughout a brain area (like a row of falling dominoes after the first one has been knocked over). Prusiner called the plaques, which multiply autocatalytically (driving themselves on) prions. He originally termed the process the amplification (replication) of a protein that had an abnormally altered structure-something that was later confused with infection.

This amplification process is considerably accelerated when an amino acid is substituted at a critical point through a mutation in the respective gene. For example, in carriers in a family, in which a certain type of encephalopathy frequently appeared, the base thymine was substituted for cytosine in the gene codon which usually encodes the amino acid leucine. The consequence is that this codon 102 gene no longer encodes leucine, but rather the amino acid proline. Proline, however, is known as a helix breaker. By 1995, 18 different mutations had been discovered in SE families in which spongiform encephalopathies or brain-softening :Onspicuously frequently occurred). Time of occurrence, degree of severity and the course of disease were dependent upon
Chapter 5
The general acceptance of the hypothesis that BSE is an epidemic (triggered by feeding animals meat and bone meal in which infectious prions can be found) means that no attention is paid to the fact that BSEs epidemiology does not correspond with the feeding of meat and bone meal at all. As an article in The Lancet shows, within Great Britain, most cases of Creutzfeldt-Jakob disease (CJD) were observed in people in northern Scotland, while most cattle with BSE were to be found in southern England, as shown in a paper printed in Nature (see diagram). But according to the mainstream BSE theory, consumption of BSE meat triggers Creutzfeldt-Jakob disease (a theory that, to stress one more time, is completely unproven), but, this could only be explained if the meat from the BSE-infected cattle from the south of England was only eaten in the north of Scotland. This, however, is practically impossible.

In 1985, a law was passed in England forcing British farmers to apply phosmet to the necks of their cattle (see diagram) Y Phosmet is what is known as an organophosphate, and the highly toxic insecticide, which causes severe neural damage, is used against warble flies. Only in Great Britain, Northern Ireland and Switzerland was phosmet used in such high concentrations-the countries where almost all BSE cases have occurred. A British organic farmer by the name of Mark Purdey noticed that his cows did not come down with BSE, ecologically-kept cows did not come down with BSE, although they had been feed meat and bone meal-but had not been treated with organophosphates.

The British government knew about these connections. And so, at the beginning of the 1990s, the law requiring phosmet application to cattle necks was repealed, since there was a likely connection between the organophosphate and the appearance of BSE. At the same time, from 1993 on, there was also a drastic reduction in BSE cases. The British BSE investigative board also admitted that organophosphate was evidently a co-factor in the onset of BSE. And it has been known for a long time that chronic organophosphate poisoning leads to a polyneuropathy [severe neural damage] , according to toxicologist Heinz Liillmann.

This was confirmed by the research results of neuroscientist Stephen Whatley, from the London Institute of Psychiatry. According to this research, financed through private donations, phosmet could be the trigger for BSE diseases. Whatley wanted to pursue the subject more thoroughly and requested additional experiment funds from governmental institutions. But the authorities rejected Whatleys application something which seems all the more baffling considering Whatleys emphasis that there is no contradictory data, that is to say there is still no scientific paper that refutes his conclusions.

In this context, why don’t all cows that are treated with organophosphates come down with BSE? One may think that the dose makes the poison. However, even if all cattle received the same toxin dose, they would not react the same way, since the cattle have individual genetic makeups. Furthermore the amount of phosmet applied by each farmer could also vary significantly. If a toxin can accelerate the outbreak of a disease (as alcohol can liver disease), then it can also be the lone cause.
If, however, it was officially verified that phosmet was a cause of BSE, compensation claims worth billions would be filed, not only against the British government, but also the insecticide manufacturers. This is certainly not a desirable outcome for the powers that be, and, so, clear connections are allowed to disappear into a fog of prions.

Incidentally, the poisoning or intoxication hypotheses are easy to test, and, in contrast to the virus or prion hypotheses, they are comfortable, meaning proof that a theory is right or wrong through toxicologic and epidemiologic verification. But unfortunately, these tests have not been carried out.

Regrettably, for about ten years, the trend has increasingly been towards the scaling down of toxicological institutes, while pharmaceutical institutes gain ever more significance. Through this, the critical aspects of toxicology (poisonous nature of medications and other chemical substances) increasingly disappear into the background, because the primary focus is researching medications.

Besides phosmet, other poisonous substances could impair the health of the cattle, such as poisoning by the heavy metal manganese. In factory farming, high amounts of manganese are fed to chickens, whereupon, by way of the processing of the chicken droppings, the heavy metal gets into the meat and bone meal and into the cattle.

Experts also refer to a possible copper deficiency, which could have attacked the cattle’s nerves. Such copper deficiencies can produce severe neurological defects and have been seen for a long time in grazing animal. Among experts, these are described as endemic ataxia.

The assumption that BSE is an epidemic in Great Britain, caused by an infectious agent called a prion in meat and bone meal has not been proven. To prove this, at least a controlled feed experiment with cattle herds would have been necessary. But this wasn’t done. According to published data on the epidemics appearance and spread, a plausible alternative hypothesis could be that a recessive genetic defect had accumulated in a few cattle herds, states Scholz. The cause would be the excessive breeding in the pursuit of the best possible efficiency in milk production, in which, as a negative result of breeding, an increased predisposition to contract BSE was coincidentally bred-in without being noticed for a long time.

But, it’s more likely that the BSE epidemic in England was precipitated by a genetically determined predisposition combined with other stresses (poisoning with insecticides or heavy metals, copper deficiency or autoimmune reaction), to which BSE-prone animals are particularly sensitive and, thus, get sick earlier. Or exposure to toxins like phosmet could be responsible. All of these theories bring us to this conclusion: BSE is not an infectious disease.

If there is no reason to assume that this disease is transmitted from animal to animal and from species to species, it makes no sense to fight it by exterminating healthy animals or entire herds. The assertion that human health is endangered by BSE derives from the unproven prions in meat and bone meal hypothesis. This claim based on a conjecture is nothing but pure speculation. vCJD (the new variant Creutzfeldt-Jakob disease) is not a new disease, but rather a once-rare diagnosis that has recently become more common (even if 1 in 5 million is still very rare).
risk of contracting vCJD through the ingestion of beef products (including the brain, declared to be the risk material) is minimal in comparison to the numerous risks of everyday life.

Chapter 6

If one believes the media, the world has repeatedly been devastated by large new epidemics over the last two decades. At the beginning of the 1980s, AIDS appeared, a few years later came hepatitis C, followed by BSE in the 1990s and by 2003, SARS (Severe Acute Respiratory Syndrome). But these new epidemics differ from epidemics of the past on one decisive point: while the plague, cholera and typhoid fever ruined whole cities, the number of those actually affected by the new epidemics is comparatively small.

According to the Robert Koch Institute, just a few hundred people die from AIDS each year in Germany. As for hepatitis C, we are still waiting for the liver cirrhosis epidemic. And the BSE epidemic has not presented most countries with a single clinical case, but rather only positively tested animals.

Although death from so-called infectious diseases is increasingly becoming a rarity (here in Germany less than 1% of all mortalities), our modern world is plagued by epidemic fear. How else could a few cases of pneumonia-and that is what it was all about with the SARS patients-invite such fear in Chinese citizens that, en masse, in large cities like Hong Kong and Singapore/they put surgical masks over their mouths? Such masks could be found on every desk in the Chinese province of Ningbo? The Industrial and Commercial Bank of China and the City Commercial Bank of China decided to stash bank notes away for 24 hours before bringing them back into circulation (in the hope that the SARS virus would waste away on the notes during this time?) and even went as far as sterilizing money by exposing it to ultraviolet light for four hours and by treating it with disinfectants.

The German sporting goods manufacturer Adidas, which produces more than half of its worldwide-sold sneakers in China, reacted with emergency response plans; even relocating production to Indonesia was considered. But first, activism on a smaller scale was practiced when a strike force distributed a leaflet of hygiene regulations to factory workers asking if all workers wore protective masks and regularly washed their hands.

German chemical giant BASF reported, meanwhile, that they had experienced an outbreak in their office, when a Chinese secretary became ill over a weekend. But luckily, all 250 employees already knew about this come Monday: after the first reports on SARS, BASF had ordered every employee to carry a card with the telephone numbers of three colleagues in their pockets, so that in case of emergency, everyone was required to call the colleagues immediately. So, over that weekend, the news had gone viral via phone lines and 20 people who worked closely with the ill secretary were ordered to stay at home. Simultaneously, the entire floor where the secretary worked was disinfected for two days, and from that time toilets were scrubbed many times daily. A BASF spokesman expressed his satisfaction: The crisis management has worked.

Lufthansa, in contrast, was completely caught off-guard by the crisis. The German airline lost more than €300 million in the first quarter of 2003 after many airplanes were grounded. And then the group announced that another 15 planes had to be quarantined bringing the total number
of grounded planes to 70. First the 11 September [with the terrorist attacks in New York], then the war in Iraq and now SARS—it’s the worst crisis in decades, said German newspaper Die Zeit about the Lufthansa situation.

In the hysteria, everyone completely overlooked the fact that people constantly contract pulmonary infections and die. Yet the World Health Organization alleges that there were just less than 800 probable SARS fatalities, in the first nine months after the outbreak of the epidemic began at the end of 2002—in China, it is worth noting, with its 1.3 billion people, as well as in Hong Kong and Taiwan. These few hundred mortalities are so few that they only make up a fraction of the pneumonia cases constantly at hand.

SARS counts among the very rare diseases, as the Deutsches Arzteblatt emphasized in April 2003. And three years later, in July 2006, they reported that the (presumably existing) SARS-Corona virus is clinically irrelevant.

Why such mass panic? Even the rock band The Rolling Stones felt compelled to avoid Hong Kong and Singapore, and the head of the University of California at Berkeley forbade hundreds of incoming Asian students from coming to the elite institute. It was even surmised that Asia’s economy and stock markets stood on the brink of collapse. And how could the tsunami catastrophe over the New Year 2004-2005 damage the Asian economy less than SARS, even though, according to WHO estimates, the giant tidal wave claimed more than 200,000 victims within a short time (easily a hundred times as many people lost their lives than those who officially died from SARS)?

The scratched windshield theory described by philosopher Paul Watzlawick in his book How Real Is Real? offers an explanation for such mass phenomena: Around the end of the 1950s, a strange epidemic broke out in the city of Seattle: increasing numbers of car owners observed that their windshields were littered with small crater-like scratches. This phenomenon gained the upper hand so quickly that President Eisenhower, at the request of Washington State Governor Rosellini, sent a group of experts from the American board of standards to clear up the mystery. According to Jackson, who later summarized the process, the committee very quickly found that, two theories about the windshields were circulating among the city’s inhabitants.

On the basis of one, the so-called Fallout theory, recently held Russian nuclear tests had contaminated the atmosphere, and the radioactive deposit caused by this had been transformed into a glass-corrosive dew in Seattle’s damp climate. The asphalt theoreticians, on the other hand, were convinced that the long stretches of freshly paved freeways, which Governor Rosellini’s ambitious roadwork program had generated, sprayed acid drops against the previously untouched windshields, also influenced by Seattle’s damp atmosphere. Instead of investigating these theories, the men from the board of standards concentrated on a much more tangible fact and found that in all of Seattle, no increase in scratched windshields could be observed.

In truth, rather, it had come to a mass phenomenon. When reports of crater scarred windshields began accumulating, more drivers began investigating their cars. Most of them did this by leaning over the glass outside and checking them up close, instead of doing it from inside and
looking through the windshield from the normal angle as usual. From this unusual perspective, pits were found which are usually there (but unnoticed) in a car that is being used. What had arisen in Seattle, then, was an epidemic not of damaged windscreen, but rather of stared-at ones. This simple explanation, however, was so deflating that the whole episode went the way of many sensation-causing reports: which the mass media first dish up as sensations, but the mundane explanations of which are kept quiet, leading to the immortalization of a state of disinformation. With SARS, doctors all over the world, likewise, suddenly looked at pulmonary infections from another angle—namely from the perspective of a dangerous new virus and a new laboratory test (SARS antibody test).

An article in the journal MMW Fortschritte der Medizin (Advances in Medicine) describes SARS suspected route of infection: On 21 February 2003, a doctor from [Chinas gigantic industrial province] Guangdong brought the virus by bus to Hong Kong, a city of seven million, where he was to attend a wedding. Already seriously ill, he booked into a hotel and allegedly infected a further seven people there, including the index patients for Canada and Vietnam [index patients are the first patients, through whom an epidemic is said to be triggered]. After his condition had rapidly deteriorated, he was taken to a hospital where he infected more patients and died ten days later. The Vietnamese index patient flew to Hanoi. There, he was treated by an Italian WHO infection specialist, Carlo Urbani, who gave the syndrome its name: Severe Acute Respiratory Syndrome (SARS). On 29 March, Urbani himself died from the infection.

And yet, every attempt had been made to protect Urbani and the patients from the evil, pathogenic microbes. As the New England Journal of Medicine (NEJM) reports, a four-hour discussion led the government to take the extraordinary steps of quarantining the Vietnam French Hospital, introducing new infection-control procedures in other hospitals, and issuing an international appeal for expert assistance. Additional specialists from the WHO and the Centers for Disease Control and Prevention (CDC) arrived on the scene, and Medecins sans Frontieres (MSF, or Doctors without Borders) responded with staff members as well as infection-control suits and kits that were previously stocked for outbreaks of Ebola virus.

The fear went so deep that, to shield Urbani from viral attacks, an isolation room was spontaneously set up, in which the expert fought SARS for 18 days in a Bangkok hospital. At the same time, guidelines for dealings with patients were published: patients should be kept in isolation and, if possible, they should lie in negative pressure rooms, rooms where the air allegedly contaminated by the virus cannot leak out.

But none of this helped; the patients died, and so did Urbani on 29 March 2003. A new causative agent—the SARS virus—was allegedly to blame. The New York Times leading medical journalist, Lawrence Altman, rushed to the scene immediately.

Shortly after Urbani’s passing, he wrote about the dangers of SARS infection: It can affect anyone who has the bad luck to be in the way of a contaminated sneeze or cough. SARS can be so explosive that scores of family members and health workers can be infected from a cough from one patient.

There is, however, no proof of this scenario. And if this were really true, then it should have
come to an exponential increase in disease cases, and the number of infected patients should have reached dizzying heights. But this did not happen, and SARS should never have been feared at any point.

A virus should also have attacked all age groups. But SARS has largely spared children-for unknown reasons, Altman remarked with surprise (without having given this important central fact any attention). Furthermore, the NEJM stated no new [SARS] cases in health care workers have been reported. In fact, no epidemic took place whatsoever-and certainly not one among health care workers. This also clearly argues against the possibility that a highly contagious virus is at work, since nurses, caregivers and doctors carry a particularly high risk of virus infection. Yet, contrary to the facts, Altman writes that, it was the quick spread of SARS to health workers that was the first major clue that a new disease had emerged.

Instead of triggering epidemic alarm, the WHO should actually have looked into the central question of why a 47-year-old doctor (Carlo Urbani) died as a result of a lung infection; something that is indeed unusual. But WHO officials suffer from virus tunnel vision, so neglected the fact that anyone who comes down with a lung infection typically has weakened immune and detoxification system. This leads to increased numbers of microbes—which consequently can end in an inflammation of the lower airways. And a whole range of substances can damage the immune system, particularly antiviral medications.

Articles on SARS in the Lancet or the NEJM show that its common to administer all sorts of antiviral and antibiotic medications to SARS patients. So, Urbani was given the full arsenal of medications-the side effects of which can very likely be lethal.

We must also consider that lung infections have never registered as epidemics. If, for example, pneumonia cases accumulate, we should ask whether an unusually high number of immune-deficient people are involved—as was the case in Philadelphia in 1976, when veterans contracted pneumonia at a meeting of the American Legion, and some died.

The United States highest virus officials, the Centers for Disease Control and Prevention (CDC), also got wind of this, and immediately sounded the alarm. A monster killer had caused the deaths of the ex-soldiers, the media cried out. The legend of veterans pneumonia caused by microbes was born.

The CDC as usual, was caught up in an infectious mania, and didn’t even think it was necessary to set up laboratory experiments so that non-microbial causes could also be traced. The discovery of a bacterium in a few victims shouldn’t lead to the automatic assumption that the microbe is the primary or sole cause of the illness. Such a bacterium could very well be a secondary invader: a bacterium that multiplies on the foundation of a weakened body. We must also keep in mind that legionella bacteria are ubiquitous in the environment, but large numbers of people (and animals) aren’t getting sick because of them. There never was any danger of an epidemic. Indeed, epidemiologic analysis of epidemic and sporadic cases has identified a variety of risk factors for the development of Legionnaires disease or for fatal infection, writes pathologist Washington Winn in the journal Clinical Microbiology Reviews after closely investigating the event. Notable among these have been cigarette smoking, advanced age, chronic lung disease
and immune suppression [weakened immune system]. It is likely that a combination of risk factors produces the highest probability of infection. Many patients, labeled as Legionnaires disease victims, are already seriously ill (with cancer, diabetes, chronic bronchitis, kidney transplants, etc.) and take immunosuppressive medications.

And so the pneumonia that struck down veterans (legionnaires) at their 1976 gathering was a bacterial infection and the veterans were easy targets because they were immunologically weakened after partying day and night (with drugs, alcohol, nicotine, or sleep deprivation, all known to weaken the immune system). Even today, there are still veteran’s disease outbreaks, which amount to nothing more than a few pneumonia cases.

The rest of the epidemic victims are test epidemic cases that crop up only because healthy people are being tested serologically (by blood test), and this test also comes out positive—which in turn can have various causes (alcohol, drugs, malnutrition, etc.).

A bacterial pneumonia can be easily determined from the blood count. As a rule, a directed antibiotic treatment is successful (even though resistance to antibiotics can increasingly be observed). Now SARS is supposed to be a viral infection, so a strong immune system will typically allow the body to fight off the virus. Alternately, the weaker the immune system, the more pronounced the viral infection. But, what weapons does mainstream medicine primarily use to fight viral pneumonia or other diseases when a virus is alleged to be the cause? Ultimately, nothing but drugs that weaken the immune system.

A good example is shingles (herpes zoster), which affects one in three people in developed countries over their lifetimes. Mainstream medicine conjectures that dormant and then sometime reactivated herpes viruses in the body (or more precisely, chickenpox viruses) are to blame for shingles. And so, for a fairly long time, it has been believed and postulated that antivirals, like bacteria-eliminating antibiotics, are an effective weapon against viruses.

One of the first antivirals, aciclovir (Zovirax), is said to fight herpes viruses and shingles. But clinical proof of this is, once again, missing. Not only do many shingles cases go away without treatment, for which reason people like to claim they react to being spoken to by wonder healers. Basically, the body’s self-healing powers (immune system responses) are at work. Additionally, placebo-controlled studies for the approval of Zovirax—as with flu remedies (Relenza, Tamiflu, etc.)—provided no proof that antivirals significantly shortened the course of disease.

It is claimed that these medications can alleviate the disease symptoms affecting the nerves, but this is a very subjective sort of diagnosis and, since it is so difficult to objectify, the pharmaceutical industry simply makes assumptions that are ultimately tailored to generating profits. Yet, antiviral substances can trigger precisely the same symptoms that they profess to fight: from anemia (iron deficiency), bone marrow damage, oversensitive skin, and breathing difficulties to defective kidney functions and liver damage (hepatitis). All of these adverse effects are noted on package inserts as well.

Additionally, as a rule, these antiviral substances are nucleoside analogues or DNA terminators, meaning that they block the genetic material (DNA) and through this are supposed to impede
virus replication. But this is not the only concept of antivirals that is tied to a hypothesis with many unproven and even contradictory factors.

The basic requirement, then, for developing active antivirals is to first know the enemy—the virus—exactly, and also knows that it is a pathogenic enemy, working alone (without accomplices like chemical toxin, stress, etc.). But with the SARS virus as well, there are justified doubts that all of these factors have been securely determined.

As we’ve said before, the most reliable proof would involve of taking blood from a patient and isolating a virus by completely purifying it (separating it from all other cell components) and then imaging it with an electron microscope. Only true virus isolation allows for the development of reliable virus tests, since biochemical determination and identification of the genes and proteins typical of a virus require it to be available in a pure culture.

The presence of foreign particles, as well as the false determination of the particle (which is possibly not even a virus at all) would be fatal, for it distorts the results upon which, ultimately, the development of virus tests are based. The consequences then include misdiagnoses, unnecessary fear of death for thousands of patients, as well as the administration of side effect-laden antiviral medications, anti-fever medicines, etc. But unfortunately, not one of the publications that have appeared to date, shows any proof of a genuine virus.

Mainstream research has hardly managed to replicate what are termed corona viruses (the so-called SARS virus is supposed to be one) in conventional cell cultures, as can be gleaned from the German Zeitung. Also, according to orthodox virus theories, the suspected SARS virus should be present in every patient—and it should not be found in healthy individuals. But no studies confirm that this is the case.

On the contrary, only very few SARS patients tested positive for the corona virus introduced as prime suspect right after SARS panic broke out, as reported in April 2003, at the first large global SARS conference in Toronto. Unfortunately, this information did not prompt orthodox medicine to ponder, even for a second, if the virus concept was really true. They’re just too busy playing with their favorite toys: the molecular biological methods—above all with PCR—and, so, think that corona viruses could be detected with them.

As always, the medical establishment is confident that SARS is a virus as well. And so, on 15 May in Nature and a month later in the Lancet, researchers in Rotterdam claimed to have delivered conclusive proof of a pathogenic SARS virus.?

436 patients, who fulfilled the case definition of SARS, were tested for the presence of a corona virus. Then, the supposed corona virus was injected into some macaque monkeys that responded not by becoming seriously ill, but rather displayed only light symptoms. Regardless, this satisfied the German Tagesspiegel enough to write that the tests on monkeys at the national influenza center at Rotterdam’s Erasmus University showed that the new corona virus triggers SARS. The in formativeness of patient sample virus tests is, in fact, highly questionable. As the World Health Organization said via a press release on 22 October 2003 (months later), there was still
no gold standard for detection of the SARS virus. In other words, the tests could not be calibrated for a specific virus.

Moreover, the presence of a corona virus was said to be confirmed in only 329 of the 436 patients who fulfilled the case definitions for SARS, according to the Lancet study. This means that even if we assume proof of the existence of the virus that causes SARS symptoms, more than 100 patients were misdiagnosed, and for no reason, suffered fears of death, were exposed to restrictive quarantine measures and were given antiviral and antibiotic medications laden with side effects.

A closer look at the monkey tests reveals another glaring weakness in these experiments. Researchers took a cellular culture which originally came from a SARS patient and further cultivated it with a complicated procedure, and administered it to four macaque monkeys through their throats, noses and under their eyelids. The animals were examined daily for the appearance of disease. On the second, found and sixth days, the monkeys were anaesthetized with ketamine and ten milliliters of blood from veins in the groin, and smears from the nose, mouth, throat and anus were taken.

Three of the monkeys became lethargic after two or three days. On the fourth day, two developed temporary rashes. One monkey had breathing difficulties, while three were plagued by non-advancing alveolar damage to both pulmonary Jobes.

The lymph nodes near the trachea and the spleen were larger than normal. The other organs in these three macaques, as well as the airway and other organs from monkey number one appeared normal under microscopic examination.

Attributing these symptoms to a specific virus, however, is impossible, since a gold standard (real detection and characterization of the virus) was missing. Apart from that, many different virus-sized particles could be captured such as different viruses or other cellular debris. Then there are the laboratory chemicals, at least traces of which still remain, and which could likewise have an effect.

Additionally, as already mentioned, the monkeys were anaesthetized with ketamine. Possible side effects of this medication in humans include increased blood pressure and heart rate, increased vascular resistance in pulmonary circulation, pulmonary edema, heightened sensory perception and intercranial pressure, increased muscle tension, dehydration, redness of skin, dreams (of the unpleasant sort) and shock conditions. During sedation or after waking up, side effects also include hallucinations, nausea, vomiting, dizziness, motor agitation and even respiratory arrest with too large a dose or too fast an administration.

These recognized human side effects can appear weaker, stronger, or altered in the monkeys, and are exactly the same symptoms observed in the monkeys (lethargy, rash, breathing difficulties, altered pulmonary tissue). But, incomprehensibly, the article doesn’t broach whether these side effects could have been caused by ketamine. H is also amazing that researchers came to their final conclusions on the basis of only four test animals, considering that the monkeys did not even continuously display the same symptoms, far less typical SARS or flu symptoms like fever
and coughing. Only one animal had breathing difficulties at all (SARS is, mind you, a pulmonary
disease).

Furthermore, in these experiments, there was no control group of animals exposed to exactly the
same (and possibly traumatic) conditions, including the physical containment and the treatments
themselves, like being anaesthetized with ketamine. Moreover, the control animals should have
received the same injections, only without the alleged virus. Only through such a control group
could the researchers truly rule out that the symptoms that appeared in the monkeys could have
been caused by something other than the alleged corona virus.

Apart from this, with antivirals, it is impossible to target specific viral genetic material (DNA).
Rather, the use of antiviral substances is equivalent to a round of machine gun shots. Through
this, the genetic material of healthy cells is always affected, meaning that their growth is
constantly impeded. Finally, antivirals work like chemotherapy in the treatment of cancer
patients, in that they are inescapably damaging to the immune system (immunosuppressive) or
even carcinogenic (cancer causing).

The reality is now that with virtually every little ache and pain, antivirals are too often prescribed
by the doctors an requested by patients. And the money rolls in for pharmaceutical groups and
doctors. But for the patients, this means that, in the long term at least, they will have to anticipate
severe damage to their health (even including cancer).

Steroids are another group of often-used and potentially problematic medications. Steroids, a
family of drugs to which cortisone belongs, are extremely effective anti-inflammatory. With this,
unpleasant symptoms like respiratory distress diminish, and doctor and patient are hopeful that
the problem has been solved. At the same time, the patient’s immune system is further weakened
due to the anti-inflammatory effects of the medication, and the course of the disease, described
as a viral infection, can in certain circumstances become worse and even have lethal
consequences.

The Kiel University Hospital had this unfavorable experience while treating so-called viral liver
inflammations. At first, laboratory values improved, but then, under cortisone therapy, severe
shingles developed.

In May 2003, the Lancet reported that many SARS patients had been treated with high doses of
cortisone and the antiviral (DNA terminator) ribavirin. But the case description, which is
probably exemplary of most SARS cases, reads like a bad horror movie in which the characters
make a serious of unfortunate choices.

The first unfortunate move was the decision to prescribe antibiotics that had no effect, because
there was no bacterial infection. Thus a worsening in health occurred. The second unfortunate
choice was to carry out an open lung biopsy. This means that a tissue sample was taken from the
lungs for test purposes. But after the operation, the patient had to be put on a respirator. This
resulted in the third unfortunate decision: high doses of antivirals and cortisone were given
intravenously. 20 days after arrival, the patient died. One can well imagine that the patient did
not die despite, but rather as a result of the therapy.
Admittedly, we could only scientifically draw such a conclusion if so-called placebo-controlled double-blind studies had been, or would be, carried out. These are tests where there are not one, but two groups of patients, from which one receives the preparation while the other gets an inactive pseudo-medication (placebo). At the same time, neither patient nor the doctors treating them knows which subject receives what (active substance or placebo), which is why they are termed double blind. Only with such placebo studies can it be said that a medication is more effective than doing nothing—or causes more damage than an inert placebo, something that is not improbable, since most medications have severe side effects. Adverse therapeutic outcomes can only be prevented through long-term placebo controlled studies. Otherwise, the doctor in charge never knows if the patient recovers, becomes ill, or even dies despite or due to the initiated measures (giving of pills, etc.). And indeed, relevant studies, including ones carried out by the American drug approval authority FDA, argue that such placebo controls (contrary to usual practice) should always be carried out.

With SARS specifically, without these placebo controls, it can by no means be ruled out that SARS patients who are only slightly ill would recover without medications like ribavirin. At the same time, they could also become completely healthy again, even though they are administered ribavirin, because their immune systems are still so sound that they can fight the drugs with toxic and immunosuppressive effects. It is just as possible that SARS patients already severely weakened with compromised immune systems are not aided at all by ribavirin, but that the diseases course is only accelerated.

A clear indication of how little sense it makes to administer antivirals, is depicted by the second case description in the Lancet study mentioned above. This paper points out that the symptoms gradually improved without treatments of ribavirin and steroids.

We come now to the therapeutic dilemma of our time. It has become noticeably more difficult for doctors to engage in therapeutic nihilism, that is, providing a severely ill patient with only life-support measures like oxygen and fluid replacement. Nowadays, in our completely overmedicated society, there is a knee-jerk reaction toward doling out drugs—from doctor and patient alike. Caution is rarely observed from either side.

Likewise, few doctors inform their patients about ways in which they can strengthen their immune systems themselves. For example, the influence of the intestinal flora [as the largest immune organ] upon health is very significant, as intestinal specialist Francisco Guarner says; it performs essential functions for the nutritional supply, the development of epithelial cells and the strength of immunity. Numerous factors have an influence upon the intestinal flora's condition—primarily nutrition.

Admittedly, doctors must also consider legal issues. They are seldom prosecuted if they have administered all sorts of medications but much more likely to be sued if they didn’t administer anything. Its generally assumed that a patient may die even though he has been treated with medical substances (even when deadly side effects are known), but it is practically never assumed that the death is due to the medical treatment. As well-known British pharmacologist Andrew Herxheimer puts it, in reference to the poisoning of AIDS patients through antiviral
medications like AZT: Damage [caused by medical drugs] is usually underrepresented in media coverage.

Of SARS it remains to say that it is a banal pneumonia from which, if unfavorably treated, large numbers of people will die. Or as Ludwig Weiss Becker, former chief of the department of internal medicine at the Kiel University Clinic, expresses it: Behind an unfortunate therapeutic outcome is often an unfortunate therapist.

With SARS, like the other alleged epidemics, virus panic superimposed everything and even though other more reasonable explanations were right under our noses. Its interesting that the first patient to trigger SARS panic came from Guangdong province in China. Here, it’s important to emphasize that in nearby Hong Kong, with its 75 million inhabitants and thousands of farms, humans and animals live extremely close together.

Yet Die Zeit spun a decidedly horrific tone when depicting living conditions in Guangdong province: The environment from which the virus presumably sprang is despicable: South China, a classic hotbed for deadly epidemics. Here, anything that has muscles and mucus membrane is eaten. Microbes easily jump from one species to another. This demands adaptation to new hosts. And this is how mutated viruses and new epidemics emerge. But this-as Die Zeit itself concedes-is pure speculation. The description also begs the question that if this were the case, how can it be that SARS first broke out in 2003, when the Chinese have lived closely together with their animals for thousands of years?

Yet, through a microbe-fixated view, another piece of the puzzle is completely suppressed which is at least as characteristic for Guangdong province as the omnipresent chickens and other animals: Guangdong is China’s largest industrial area, acting as a sort of global workshop with its textile, toy and microchip factories. This region is the hub for China’s exponential global economic growth. It’s a paradise for politicians, corporate investors and multinational corporations, but this is exactly why the area is extremely polluted. Garbage lies everywhere; above all high-tech waste. Computers, mobile phones and the Internet are supposed to help poor countries achieve the kind of prosperity Western nations enjoy. But the age of information has caused many problems for developing countries, including masses of electronic scrap and toxic waste. Up to 80% of electronic waste accumulated in the USA (10 million computers per year alone) is not disposed of in the land of boundless possibilities, but rather, through a series of dealers, the high-tech waste is sold to the best-paying customers on the international market. At the end of this chain, as the study Exporting Harm: The High-Tech Trashing of Asia shows, are the poor in India, Pakistan and China—and there, above all, the people in Guangdong.

For $1.50 a day, locals disassemble computers, monitors and printers with their bare hands, endangering both their own health and the environment. The export of E-trash is the high-tech revolutions dirty secret, says Jim Puckett of Basel Action Network, one of the study’s co-authors. A short time ago, the import of high-tech junk was officially banned. But the waste makes it to China, be it because the regulatory authorities are simply overwhelmed or because corruption makes import possible.

One of the places where the authors did their research was Guiyu in Guangdong, which
developed from a rural spot into a booming centre of e-waste processing since the mid-1990s. There, workers empty toner cartridges from laser printers the whole day long without protective masks, breathing in fine carbon dust. Others, mostly women and girls, dip circuit boards into baths of liquid lead to separate and collect the soldering materials with which the memory chips and processors are attached to the plates.

Unprotected, they are exposed to toxic fumes. While the plastic plates are simply burned up, the chips and processors are put in acid baths, to extract their gold. Here as well, poisonous fumes are generated, and the unusable leftover acids are just dumped into the river. A lot of garbage is simply burned up or dumped onto rice fields, irrigation facilities or into waterways. The bodies of water and groundwater around Guiys have become so contaminated that drinking water has to be brought in daily from other cities.

Many heavy metals and other highly toxic substances are suspected to cause serious health problems, including cancer and neural damage. According to studies, the high level of contamination [in Guangdong] caused by unsafe electronics disposal is a potentially serious threat to workers and to public health, said Arnold Schecter, a professor of environmental sciences at the University of Texas School of Public Health. I think we’re fooling ourselves. We think we’re doing the right thing by recycling, but we’re harming people in less developed countries.

Chapter 7
If one believes the media reports about avian flu, the world will be afflicted by a global epidemic—a so-called pandemic—in the near future, triggered by a mutation of an avian flu virus with the mysterious and ominous-sounding name H5N1. In the weekly newspaper Die Zeit in late summer 2005, we read with shudders this FrontPage headline: Death on silent wings—the bird flu is approaching. And, as if the point was to create the title for the sequel to the Hollywood shocker Outbreak, in which actor Dustin Hoffman is on the hunt for a deadly virus: H5N1 plays Blitzkrieg [lightning war]; impending attack of the killer ducks.

Der Spiegel quoted David Nabarro, named the UN chief coordinator in the battle against avian flu in September 2005: A new flu pandemic can break out any moment—and it can kill up to 150 million people. Reinhard Kurth, director of Berlins Robert Koch Institute, didn’t want to be outdone by Nabarro and, in an interview with the Frankfurter Allgemeine Zeitung he warned that, an epidemic potentially threatens all six billion people.

A more detailed inspection of media reporting on the subject shows one report or another that actually downplayed the virus panic. The Canadian news magazine MacLean’s (the country’s equivalent to Time in the USA) printed an article headlined: Forget SARS, West Nile, Ebola, and Avian Flu [H5N1 ]—The Real Epidemic Is Fear.

Marc Siegel, professor of medicine at New York University and author of the 2005 book False Alarm: The Truth About the Epidemic of Fear, presented his critique of the fear mongering climate in several media simultaneously, including the Ottawa Citizen, the Canadian capitals most significant daily newspaper, the Los Angeles Times/ and USA Today.
In German-speaking regions, Freitag, Berliner Republik, and Journalist were among the publications, that ventured to be critical; and the Swiss Weltwoche wrote: Only when the last chicken has laughed itself to death will you see that horror reports are more contagious than BSE, SARS and HSN 1.

Sadly, the few levelheaded voices got completely lost in the tidal wave of HSN1 virus-manic reports. Under this apocalyptic cloud, there were few attempts to get to the facts, which should have happened from the beginning. Are the warnings churned out by newspapers, magazines and television stations and sold to a global public as the final conclusions of truth, backed up by scientific proof? Quite evidently not.

The scientists and their lobbyists seem more interested in acting as media celebrities. These mainstream virus experts do their rounds in newspapers and on television, creating a guise of legitimacy. The media repeats exactly what these so-called experts want to hear without asking for evidence. We discovered this after getting in touch with various publications asking the following questions:

1. Is an independent study available to you, which proves that the so-called HSN1 virus exists?
2. If there’s proof of the virus existence, is an independent study available to you, which proves that the HSN1 virus has pathogenic effects on animals?
3. Does sound evidence exist that rules out other factors (chemical toxins, foreign proteins, stress, etc.) as causes of the avian disease?
4. Is an independent study available to you, which proves that HSN1 can jump to the human species and can trigger a pandemic with many millions of deaths?

Even opinion leaders like the Spiegel, Frankfurter Allgemeine Zeitung or the Frankfurter Allgemeine Sonntagszeitung, however, could not name a single study. Die Zeit merely wrote: All primary sources [studies] can easily be looked up using [the scientific databanks] DIMDI or Pubmed, and can then be ordered through [the document delivery service] Subito. Experts from the Robert Koch Institute, for example, or the National Research Center for Viral Diseases in Riems are open to questions from any journalist. And the relevant CDC and WHO publications are freely accessible.

In response, we told Die Zeit that the research methods they had mentioned were very familiar to us and we were only asking them kindly to name what we had requested: concrete studies. But there was no answer.

Many people will be bewildered by this information. Can the public really assume that the mainstream media (which pitches itself as a watchdog of political and economic powers-that-be) critically filters the statements of the medical industry and other interest groups-and do not simply function as megaphones, strengthening the industry’s advertising messages?

The HSN1 hysteria made it clear that the media hangs on the words and opinions of the establishment, perhaps most especially regarding medical science. This was also shown by the paper Bitter Pill, which appeared in, arguably, America’s most significant media journal, the
Columbia Journalism Review (CJR) in the summer of 2005. It describes in detail with numerous examples, how the medical industry uses the media to play out their modern marketing script: first by depicting scenarios of horror, creating the desire and demand for a remedy (typically in drug form)-and finally, the miracle substances come to the rescue, providing the pharmaceutical companies and their researchers high profits.

Not only do journalists naively trust the leading medical officials. The news media too often seem more interested in hype and hope than in critically appraising new drugs on behalf of the public, as CJR writer Trudy Lieberman outlines. [And] the problem has grown dramatically in recent years as direct-to-consumer advertising has increased, delivering ever-higher ad revenues to the nations media.

In 1980, Big Pharma spent just $2 million in the USA on marketing and advertisements—but by 2004, this sum had swelled to several billions of dollars per year. And instead of standing apart from the phenomenon and earning the public’s trust, writes Lieberman, the press too often is caught up in the same drug-industry marketing web that also ensnares doctors, academic researchers, even the FDA, leaving the public without a reliable watchdog.

Like the media, the German Federal Consumer Protection Ministry, government ministries of countries like the USA, Canada and France, and the World Health Organization firmly assume that HSN1 is a highly contagious virus. Or as Anthony Fauci (director of the powerful American National Institute of Allergy and Infectious Diseases and one of the eminent figures in American viral science who had already contributed decisively to the establishment of the HIV = AIDS dogma) put it: HSN1 is a time bomb waiting to go off. Later, in September 2006, the World Health Organization and the World Bank did a cost calculation, announcing that an avian flu pandemic could cost the world $2 trillion.

These are words with explosive force, which begs the question: Can these authorities, upon whom the media relies in its HSN1 reports, back up their statements about an avian flu pandemic linked to such wide-reaching consequences with hard facts?

We sent the German National Consumer Protection Ministry (BMVEL) our four central questions, whereupon we received the following answer: You are asking about very specific issues, which, at present, the Ministry—we ask for your understanding—cannot answer as quickly as would be necessary for your research. We wrote back that we had plenty of time, and would only like to know when we could expect an answer.

At the same time, we pointed out that the Ministry should actually have been compelled to have evidence at hand. Otherwise, it could hardly be justified for the Ministry to appear before the public with statements expressing no doubt that HSN1 exists, is highly contagious, pathogenic (disease causing) and so on. Nor, without evidence at hand, should they have been spending millions of tax dollars on the battle against HSN1. But the Ministry could not name any studies and simply insisted: Your requests for evidence of the pathogenicity and pandemic potential of the HSN1 virus and the studies that prove this can only be answered by the experts at the Robert Koch Institute and the Friedrich-Loeffler-Institute.

We then turned to the Friedrich-Loeffler-Institute (FLI), which, according to the Consumer
Protection Ministry, was in possession of pure H5N1 viral cultures. As a response, the FLI sent four studies, published in the well-known American scientific journals Proceedings of the National Academy of Sciences, Science, Journal of Virology, and Emerging Infectious Diseases. But neither these papers, nor the paper by Subbarao et al (which appeared in Science in 1998) cited in the Emerging Infectious Diseases paper claiming that H5N1 had been found in a human for the first time in 1997, yield actual proof of H5N1 (and these papers did not contain evidence for our other three questions either).

For avian flu, like the other alleged super viruses, biomedical research simply pulled its magic wand—the biochemical replication technique PCR (polymerase chain reaction)—out of its bag of tricks. Through PCR they claimed that the H5N1 virus genetic material is replicated, and through this the virus had been detected.

But in fact, PCR, as Terence Brown maintains in his standard work Genomes, cannot be used to detect viruses that have not been decoded (sequenced) beforehand. And a complete decoding of H5N1’s genetic material, which is necessary in order to know what exactly is being replicated using PCR, has never taken place. In any case, nobody could send us such a study (details on this topic can be read in: Engelbrecht, Torsten; Crowe, David, Avian Flu Virus H5N1 : No Proof for Existence, Pathogenicity, or Pandemic Potential; Non-H5N1 Causation Omitted, Medical Hypotheses.

So, once again, there is evidently no electron micrograph of a pure and fully characterized H5N1 virus, either. There were pictures of alleged H5N1 viruses printed in media sources, but these were computer animations or completely normal cellular components that had been artificially produced in a test-tube (which is easily recognizable to any molecular biologist). The layperson can verify this by requesting a specialist peer reviewed publication in which H5N1 is illustrated and described in all the glory of its genetic information from the authorities in question, like the American CDC or the FLI. If anyone receives such a paper, please forward it on to us. Since H5N1 has never been seen, avian flu antibody tests—like SARS, hepatitis C, HN and modern viral science in general—attempt to prove the existence of the deadly enemy in an indirect way. The claim is that an infected individual has very special antibodies directed against this particular H5N1 virus. But such highly specialized antibody tests could only be constructed if it were clear exactly what the tests reacted to when they came out positive or negative. But here we’ve come full circle, for this would only be possible if tests were calibrated for an H5N1 virus, but there is no proof that such a thing exists.

Because of this, it is impossible to say that H5N1 can cause disease. Orthodox researchers say that the pathogenicity of viruses like H5N1 can be proven in the laboratory by inoculating it into fertilized eggs or animals that have already seen the light of day (the neon light of the test laboratory). But, a look at the publications in which the experiments are described shows no proof of pathogenicity.

In the laboratory experiment which the FLI presented as evidence of H5N1’s pathogenicity, large amounts of the test extract (which may have contained all sorts of cellular components and other
potentially damaging material) was injected into ducks windpipes, nasal cavities, eyes and throats for days. All the damage and destruction this extract caused was then passed off as the result of an HSN1 virus.

Such details do not interest the mainstream media. They keep playing their game of blown up horror stories and simultaneously credit scientists for their reports. In mid-January 2006, Spiegel Online jumped on the mega-story that HSN1 was said to have swooped in and killed three Turkish children; the headline read: HSN1 virus adapts to humans. In the story, writers referred to WHO scientists who claimed to have discovered a genetic alteration into a virus that could also become dangerous for humans during their analysis of the young victims.

But that this mutation had already adapted to humans, as the headline suggests, is not provable, as the Spiegel admits in the body of the article: It is still too early to estimate decisively whether the mutations are dangerous [for humans] as the WHO declared. The WHO experiments were not published in any peer reviewed medical journals, so we inquired repeatedly at the WHO, requesting they send us papers on these experiments or simply tell us their titles so we could examine them for ourselves. But the World Health Organization did not respond.

As with SARS, BSE, hepatitis C and HIV, it is necessary with HSN1 to move away from the fixation on viruses. For decades, we have been able to observe how animals in industrial poultry farming become sick: their combs turn blue, their egg production is reduced, or their feathers become dull.

The FLI, Germany’s national institute of animal health and national avian flu reference laboratory, describes the symptoms that appear in birds in its information pamphlet Classical avian influenza—a highly pathogenic form of avian influenza [highly contagious form of bird flu]: Animals are apathetic, have dull, ruffled feather coats, and high fevers and reject feed and water. Many exhibit breathing difficulties, sneezing, and have discharge from eyes and beak. They develop watery slimy, greenish diarrhea and sometimes exhibit disruptions to the central nervous system (abnormal posture of the head). Water deposits (edemas) can appear on the head, wattle, comb and feet can turn purple through congestion or internal bleeding. Egg production is interrupted, and eggs that are produced have thin and deformed shells, or no hard shells at all (wind eggs). In chickens and turkeys, mortality rates are very high. Ducks and geese don’t get sick as easily, and the disease does not always lead to death. Sometimes they suffer from an intestinal infection, which is outwardly almost unnoticeable, or else display central nervous disruptions.

For years, a virus has been claimed as the sole cause of these disease phenomena, something which the FLI also takes for granted, writing in its information flyer on Classical Avian Influenza: How is avian influenza transmitted and spread?

Diseased animals eliminate masses of the infectious agent with feces and mucous or fluid from the beak and eyes. Other animals become infected through direct contact by breathing in or pecking at material containing the virus.

By presenting as irrefutable fact something that has not been scientifically proven (no proof of
virus existence, no proof of the transmittable or infectious mechanism), viral research commits a most basic error. It neglects its highest duty, namely, to investigate if factors other than microbes cause or at least are contributing causes of the disease in birds. In fact, these factors are characteristic of factory farming:

- Heavy psychological stress resulting from extremely close crowding in the cages and mass stabling with no natural sunlight
- Denatured industrial feed, including already spoiled feed
- Distortion of animal bodies as a result of over breeding for certain desired physical characteristics
- Preventive administration of all sorts of side effects-inducing medications (antibiotics, vaccines, etc.), even to chicks

You don’t have to be a scientist to suspect that animals exposed to these unnatural conditions for a lifetime can become ill. A major offender, as studies show, is high-performance breeding, which pumps the animals up, while simultaneously degenerating them in many physical areas, so that the livestock become ill almost independent of the husbandry system. This breeding is so extreme that many species would not be able to manage in natural husbandry conditions. Imagine trying to keep a high-performance cow with a super-sized udder that produces 8,000 liters of milk per year in a meadow without giving her concentrated feed? It wouldn’t work at all. No less degenerate is the situation with poultry. Eight week-old chickens today are equipped with seven times the chest musculature as nine-week-old chickens 25 years ago, as John Robbins describes the gruesome reality of factory farming in his book The Food Revolution.

Numerous animals also suffer from skin diseases, chemical burns (hock burns), skeletal problems and paralysis. In the European Union alone, many tens of millions of hens in the mass pens are affected by lameness, which can be associated with severe pain caused by abnormal skeletal development and bone diseases (in many large facilities, half of the animals are affected by skeletal growth problems). These lame animals spend up to 86% of their time lying down, so that they sometimes cannot reach the drinking water container for days at a time. Countless hens are also tormented by heart problems; many animals die of sudden cardiac arrest (sudden death syndrome). Experts estimate that in the EU, around 90 million chickens per year die as a result of heart defects, which can primarily be linked back to over breeding—the heart simply cannot keep up with the extremely stimulated body growth. Additionally, the air in the gigantic halls where the chickens are kept can be so full of dust and biting ammonia that the animal’s eyes, throats or lungs begin to turn, resulting in diseases, collapsed lungs and a weakened immune system.

Even assuming that a virus with pathogenic potential is somehow a culprit, it is sciences duty to clarify the roles played by other possible disease-causing factors (like factory farming itself). And indeed, the FLI admits that the clinical pictures that the flu virus produces in the birds are similar to other clinical pictures. Altogether, the FLI lists eight similar clinical pictures—so-called differential diagnoses. But unfortunately, they only take these into consideration when they can’t nab an influenza virus as culprit. Furthermore, the first seven spots on this eight-point list are diseases which mainstream medicine firmly assumes are caused by microbes (like so-called pneumo viruses or microbes believed to be the primary/single cause of infectious bronchitis)—
and only at the very end, in eighth place, are poisonings mentioned, with no further detailed explanation.

Thus, before checking if the animals disease symptoms have been caused by poisoning with medications, spoiled feed, chemicals like ammonia and so on, examiners first look to see if seven different infectious agents triggered disease. And if they think they have apprehended such a microorganism, they simply stop searching for other potential toxins. Poultry farm inspectors fall in step with this virus fixation. In 2003, when avian flu panic broke out in Holland, samples from diseased animals were sent in, but no samples of feed, water, litter or indoor air.

The study could hardly have been more single-mindedly directed at microbes. The FLI did tell us that it had investigated if factors other than the alleged H5N1 virus could have led to the illnesses among Chinese wild birds (believed to trigger the 2005 avian flu and eventually exterminated). But none of the studies we received from the FLI look at any causes beyond H5N1—not even from the paper that is explicitly said to support the FLIs statements: Role of domestic ducks in the propagation and biological evolution of highly pathogenic H5N1 influenza viruses in Asia, published in Proceedings of the National Academy of Sciences, 26 July 2005.

Obviously no further research was done after they thought they had discovered a virus with the assistance of indirect detection procedures (PCR and antibody tests). But, as already mentioned, these indirect proof procedures do not confirm the existence of a certain virus. And they certainly don’t deliver evidence that this is a disease-causing virus.

Many experts like veterinarians and also small poultry breeders, meanwhile, continue calling attention to the fact that the so-called avian flu is by no means solely a phenomenon of factory farming, or that keeping laying hens in cages actually makes them less susceptible to disease than if they were kept in free range husbandry. But under closer observation, these clues do not add up.

The caged animals must battle substantial health problems and death rates. Even in the so-called enhanced cages, walking, running, fluttering and flying are just as impossible as in conventional cages, which are the size of a standard sheet of paper. And a consequence of lack of movement is a reduced bone stability, osteoporosis, from which skeletal anomalies and painful broken bones can result, states Ute Knierim, professor of Applied Farm Animal Ethology and Animal-Fair Husbandry in the Department of Ecological Agricultural Science at the University of Kassel.

Here, disease is all too hastily equated with microbial or viral infection. But whether, for instance, free-range animals have also really become sick because of a virus or because of other factors must first be closely investigated in detail. In any case, when requests are made for concrete studies, no studies are named. The typical response is, Oh, everybody knows that, or that the conclusion was made through personal experience.

Personal experience is certainly useful and here there is evidence to show that modern production methods make animals sick. We learn from our elders, who grew up on chicken farms in the 1920s and 1930s, a time when the birds could run around and peck away in a much more natural environment and were generally fed very natural food (com, fresh vegetables, etc.).
These birds never had a bluish comb discoloration or dull feathers. So, it’s reasonable to conclude that the type of a husbandry is important, and perhaps even the deciding factor in the animal’s health.

At first glance, modern free-range husbandry might sound like a good thing, but it is all too many times anything but—rather it also constitutes a sort of factory farming. Often, many thousands of chickens share a limited grass surface; up to ten chickens per square meter. Typically, larger problems occur in larger flocks, according to Ute Knierim. We must remember, though, that these conditions don’t necessarily cause viruses. For example, an investigation by the Research Institute for Organic Farming (FiBL) shows that with the increase in flock sizes, feather picking, which compromises health, also increased. Feather-picking is a serious problem that still has to be solved in order to establish whether it’s fair to keep laying hens in larger flocks, says Helen Hirt, animal breeding and husbandry expert at the FiBL.

It’s no coincidence that various livestock husbandry facilities have introduced an upper limit on flock sizes. Particularly as studies show that laying hens from large flocks use the important green space less than hens in small flocks. Why this is the case is not absolutely clear, but it has been observed that the green surface is unevenly used by the animals, which in turn leads to an overuse of the grass close to the coop, and in many cases to the turfs destruction and consequent over fertilization of the soil in this area. For animals constantly pecking at the ground, this can present a large problem. According to Hirt, the question of how turf can be kept intact is one of the most important for laying hens with pasture.

One possible way to make chickens spread out is to erect a shelter where the animals can take their dust baths. Our domestic chickens are descended from Bankiva chickens that lived in forests offering shade and places for retreat. And the need to be in an environment offering covered areas continues with our domestic chickens, says Hirt. Indeed, investigations show that chickens do spread out better over the green surface when sand-bath shelters are made available to them.

These short explanations clearly show that poultry breeding appropriate to each species that encourages robust health is a difficult undertaking. But the primary goals of many livestock owners are not maximum profits but also the animals health.

Unfortunately, all too often, they do not have sufficient professional knowledge to guarantee that their birds stay healthy. So, just like in human medicine, the animals are hasty and frivolously administered highly toxic medications, and are fed all sorts of things, from artificial industrial feed to human favorites like popcorn or chocolate—things to which the animals are certainly not genetically adapted. All of this is really worth bearing in mind, as is the practice of regularly giving young chicks numerous vaccines (see also the Epilogue: Side Effect-Free Alternatives to Medications and Vaccinations, at the end of this book).

Besides general know-how, the smaller rural structures, in which owners take care of the animals themselves and thus may have better training and more interest in the animal’s well-being, probably also play a part in the realization of considerably better results, summarizes Knierim.
But individual factors, like access to a cold scratching shed and the origin of the hens, evidently have strong influence upon the success of an alternative way of keeping laying hens.

Moreover, studies have shown that an artificially triggered laying interruption has benefits. This usually occurs through substantial light reduction and feed restriction. At first, it can put considerable strain on the animals. But at the end of the laying pause it was shown that both the strength of the eggshells and the quality of the proteins had significantly improved. The weight of the eggs had also sharply increased and markedly less feather damage was observed in the animals at the end of the laying pause.

Chickens-like all animals used in agriculture—are natural beings, reminds Hans-Ulrich Huber from the Swiss animal protection organization STS. For this reason, they should not spend their lives exclusively in coops, but should also experience sun, earth, plants, air and light. This corresponds to their inherent needs and boosts their health! For wherever the sun doesn’t reach, comes the vet.

The HSNI scare, which affected Germany via the island of Riigen in the Baltic Sea, is also no more than an artificially produced test epidemic, in which dead birds are searched for, found, and collected by the German armed forces and tested by so-called epidemic experts. That the occasional bird reacts positively to the tests is no reason to panic, since nobody can precisely say what causes a positive or negative reaction to the tests. In any case, that it is an evil HSNI virus is, as outlined, anything but proven.

Another striking fact these scientists chose to overlook is that only a fraction of dead birds discovered react positively to the HSNI tests. At this point, health officials should have asked what had caused the death of all the HSNI negative birds. And did more birds die that year than the previous year? Or did they search more for dead birds? These are self-evident questions that the scientists, the politicians and the media chose not to ask. A rare exception appeared is the Tageszeitung, which quoted ornithologist Wolfgang Fiedler of the Max-Planck-Institute: Despite bird flu, avian mortality rates on Riigen have not to date been higher than in other years. An even more difficult question to answer is why the assembled experts chose not to carry out proper research. They certainly didn’t look for the source of the (purported) avian flu infection on Riigen. How on earth could Rugen’s swans become infected with the dangerous HSNI virus? asks The Spiegel, referring to reports from the Associated Press and the German Press Agency. Researchers have a mystery before them. For the birds had wintered in Germany—and as a result didn’t come from the alleged epidemic areas. The bird population on Riigen, as ornithologists reported, is basically isolated in winter, something which clearly speaks against the possibility that the swans somewhere became infected with an HSNI virus.

But scientific and political powers ignore every doubt, pass over every inconsistency and simply stick to this: HSNI is the deadly enemy. They’re not interested in proof-speculation is enough. And so the allegations continue to pose as truths: that HSNI came out of the Far East, where, since late 2003, it is said to have caused several outbreaks of avian influenza in various Southeast Asian countries, including Korea, Indonesia, Vietnam, Japan, Thailand, Cambodia, China (including Hong Kong), Laos and Malaysia—and by mid-2005, more than 100 million animals had died. Mind you, even according to official statements, only a fraction of the deaths
are accounted for by HSN 1. By far the largest proportion of the birds died as a result of the mass-exterminations prompted by the virus-panicked authorities.

The prevailing practice is as follows: a chicken (or another bird) is singled out because it lays fewer eggs or gets a blue comb; its then sent to virus hunters and tests positive for HSN 1; and an epidemic of panic breaks out among humans! Consequently, all chickens in close proximity are gassed to death. And ultimately, statistics show that these 100 million chickens were killed by the avian flu virus HSN 1, further fanning the flames of panic.

It would be a mistake to assume that these gassings are the product of some cruel Third World practice. In early 2003, Dutch officials on the border to the German state of North Rhine-Westfalia (NRW) reported that health problems with a very high death rate had been observed on six poultry farms.

This immediately triggered epidemic hysteria. The next day (a Saturday), no-go zones within a radius of 10 kilometers of the affected farms were erected and poultry shows were prohibited. Additionally, the Netherlands banned exports of poultry and eggs. On the same day, the government of NRW issued an import and export ban on poultry products coming from their EU neighbor. Dozens of operations that had delivered chickens or feed from the Netherlands in the days before were put under official observation. Immediately, the search for a virus began using indirect test procedures—and look at that! The very next day, came the announcement that a highly pathogenic virus of the type H7N7 had been found.

Over the following four months, 26 million chickens in the Netherlands, around 2.5 million in Belgium, and approximately 100,000 in NRW were gassed with carbon dioxide, poisoned by lethal injection, electrocuted or manually slaughtered, according to Hans Tolzin, editor of the German vaccination publication Impf-Report, who did extensive analysis of the event.

Yet the media jumped on the virus bandwagon. German Stern magazine falsely reported, approximately 30 million animals perished from the bird flu in the Netherlands. And the weekly newspaper Die Zeit said that, The impending attack of the killer ducks could destroy the existence of German chicken breeders. A bird flu like in 2003 is imminent. Then, millions of chickens lost their Jives in the Netherlands and in the town of Viersen on the lower Rhine—which likewise suggests that a virus had wiped out the birds. But these media claims are ridiculous because the virus was only found in single animals (or more precisely, a H7N7 virus was said to be identified in individual animals). In the end, million birds died from another all-too human strain of virus mania.

Zeit and Stern rode the waves of public virus panic—in this case, giant killer waves. The killings ultimately swelled to such a size that the capacity of extermination and cremation facilities was no longer sufficient. A state of emergency was imposed on Dutch communities, and they were barricaded off by the military. When a few diseased chickens were found on a farm, the farms complete chicken stock was preventively exterminated, along with the stocks of surrounding farms. The economic damage in the Netherlands alone cost more than € 100 million.

But the existence—or even the dangerousness—of this so-called H7N7 virus was likewise never
proven. And while there was, once again, reason enough to look for other causes (the effects of
factory farming on the animals health, for example), the authorities declared H7N7 the enemy-
and eureka!-another epidemic was born.

The epidemic was announced on 23 February 2003, and since then, I have collected and
evaluated all accessible press releases and official reports, says Tolzin. But there was only a
single report with researchable details, from which it emerged that other causes besides the avian
influenza had been taken into consideration. But even this report, which was penned by the
Dutch Agriculture Minister Veerman on 3 March, was never mentioned again.

Everyone was clucking about a virus in the Canadian province of British Columbia, when, in
November 2005, a single duck was found and using modern indirect molecular biological proof
procedures, the avian flu virus H7N was allegedly detected. The animal, as was officially
reported, had only a mild form of this virus type, which produces no or only mild disease
symptoms. That is to say, the duck was not sick.

According to Canadian authorities, it was not the virus circulating in Asia [HSN 1 i]. There is no
new threat to human health. However, preventively, the authorities not only killed the single
duck, they immediately slaughtered a further 56,000 healthy duck and geese. Yet international
statutes certainly do not necessitate taking such drastic measures of killing entire flocks of birds,
if, as was presumed in this case, that only a low pathogenic virus is in the game.

There’s paranoia, there’s politics and there are perceptions that come into play here that cause
people to do things for other reasons than what you would call true science, says David
Halvorson, an avian flu expert at the University of Minnesota. I tend to look at it from the
scientific perspective that [the killings are] a waste of animals lives.

The haste, with which authorities and media hit the virus panic button by exclusively suspecting
a virus instead of considering a wide spectrum of possible causes from the beginning, is also
shown by the incident of the geese deaths in the German province of Rhineland-Palatinate in
October 2005. A boy had found the dead greylag geese and informed the police. The dead geese
were floating in the pond, described a police spokesman in Koblenz. And some animals perished
from severe cramps before the eyes of the action force.

In response, the dead birds were collected in cases by firemen wearing special protective suits,
and brought into the state investigations office, which immediately prompted the media to stir up
the H5N1 panic. Avian flu suspicion: mysterious deaths of geese near Koblenz and Gottingen
have strengthened fears of an avian flu outbreak in Germany, reported the news channel N24. In
turn, this prompted Jürgen Trittin, then German Minister of the Environment, to announce that
he would initiate resolute counter measures, in case the dangerous H5N 1 virus was detected in
these birds.

It turned out that the birds had been poisoned, as the regional inspection office reported. Its
president, Stefan Bent, said that a rat poison had been detected in the stomachs of twelve of the
22 cadavers. The toxin phosphide had clearly caused the deaths of the Wild geese. And even if
the presence of the rodent poison phosphide had only been proven in twelve stomachs, Bent said
it could be assumed that all the animals died from it. The toxic caused abnormal alterations in the
inner organs of the animals, like round hemorrhages on gastric mucous membrane and increased
fluid in the lungs.

Rodent poison, mind you, is not only used in Germany. In a comprehensive 2003 report, the
Japanese Agriculture Ministry tried to trace the progressive routes of flu virus outbreaks in birds
in factory farms: Poison bait type rodent poison was used during the summer and was applied
continually [against mice and other wild animals] replenished when required.

These incidents show how important it is to look at the full picture when researching possible
causes. Such a broad-spectrum viewpoint would also have been most advisable in the case of the
many thousand wild birds found dead near Chinas largest salt-water lake, the Qinghai Hu,
between May and July 2005. It reignited global panic over avian flu, because epidemic hunters,
politicians and the media immediately, and with rock-solid conviction, put their bets on an HSN1
outbreak. Once again, many other causes come into question. Pollution, for instance, presents a
huge problem in China, as in most developing countries, not least because of the chemical
industry, one of the country’s fastest-growing economic industries.

In the first half of 2005, production value rose by 27% compared to the previous year. Recently,
many new chemical factories have sprung from the ground. These facilities also produce
products for developed countries, in which dangerous chemical factories are not welcome, as
Greenpeace expert Kevin May explains. Factories are often built on rivers, since water is needed
for the production process. And of course, this is dangerous for inhabitants who drink the water,
says May.

Even without major accidents, factories in China present a danger to people’s health and the
health of the environment-including wild animals. 70% of all Chinese rivers are polluted,
because the industry directs its waste into the waterways, according to official statements.

There is also no concrete proof that water birds at Qinghai that may have been infected with such
a pathogenic strain and have survived, will migrate and be capable of transmitting the virus to
other species of birds, animals or humans, according to Wetlands International, a global nature
protection organization linked with many institutions. One of its partners is the UN
Environmental Program (UNEP), a group that deployed an expert task force composed of
representatives from nine different organizations in late 2005, as it was held to be urgently
necessary to get to the bottom of the avian flu hype. The knowledge concerning central aspects
of the bird’s deaths, it was said-including the question of how the virus is transmitted from wild
birds to domestic animals-could by no means be considered certain.

The UNEP warned of growing hysteria. Additionally, they criticized the one-eyed approach in
the media which grossly oversimplifies the causes and the methods needed to counter-act in the
interests of human and animal health. The media, so it was said, should provide more balanced
reports focusing on the facts. Simultaneously, the Task Force calls for much greater emphasis by
governments and local authorities on combating the role of factory farming, writes William
Karesh, member of the task force and director of the Wildlife Conservation Society’s Field
Veterinary Program.
Most striking is that even the medically very orthodox WHO admits, the role of migratory birds in the spread of highly pathogenic avian influenza is not fully understood. Wild waterfowl are considered the natural reservoir of all influenza A viruses. They have probably carried influenza viruses, with no apparent harm, for centuries. But, if even from mainstream sciences perspective, wild birds rarely or never become ill or die from avian flu viruses, this must have prompted even more curiosity to research other non-viral causes. Why would the wild animals get sick or even die from viruses at the beginning of the 21st century when they have lived in peaceful coexistence for millennia?

According to official statements, H5N1 caused the deaths of 153 people from the end of 2003 until November 2006. But if we study the reports on the deceased closely, there is no evidence for the theory that H5N1 was the killer. At the same time, the reports also allow completely different possibilities appear as plausible explanations. For example, that some of the victims were suffering from cold symptoms of an unknown source and then simply had the bad luck to fall into the hands of medical professionals who turned out to be H5N1 hunters.

Immediately, doctors prescribed prodigious amounts of medications in order to wipe out an imaginary virus—but in truth, it was never shown that these medications could combat the alleged virus. On the contrary, it is a fact that the medications are highly toxic, for which reason it is completely possible that the doctor’s only helped snuff out the weakened patients lives.

The Friedrich-Loeffler-Institute sent us a paper that claims to show that H5N1 has pathogenic effects in humans. The report features just one six-year-old boy. The child was suffering from a lung infection, and an aspergillus infection was also diagnosed. Whereupon the little patient was treated with antimicrobial medications that can seriously damage the immune system, as well as with the antiviral medication Tamiflu (oseltamivir), that has even been connected with fatalities (more on Tarniflu below). The boy’s fate?

The patients died during the late phase of the disease after intensive treatment with antiviral drugs. Methylprednisolone had also been prescribed to the boy a few days before he died, 17 days after initial diagnosis. The steroid is known to weaken the immune system and should not be used in the presence of a severe bacterial, viral or fungal infection (as was the case with the boy. Additionally, the report admits that, The multiorgan dysfunction observed in human H5N1 disease, despite the apparent confinement of infection to the lungs, has remained an enigma. That is to say, what is termed H5N1 could not be detected in various diseased organs at all, which researchers simply shrugged off as an enigma instead of calling it what it clearly was and is: evidence that the established H5N1 theories make no sense.

In the 1998 Science paper by Subbarao et all (also cited in the article in Emerging Infectious Diseases), a three-year-old boy was described who was healthy until, on 9 May 1997, when airway problems appeared, indicating a cold. Doctors responded by giving him Aspirin and a broad antibiotic coverage, whereupon the child developed Reyes syndrome. This is a severe disease associated with nausea, personality disorders and comas that can seriously damage organs like the brain and the liver and in many cases ends in death. Just like the other boy, he
died on 21 May. An H5N1 virus was cited as his cause of death, but here as well, evidence of H5N1 was not provided.

The medical authorities didn’t even confirm if the boy had ever been in contact with birds. Apart from this, studies suggest that Aspirin can trigger the Reyes syndrome that was also diagnosed in the boy. The National Reyes Syndrome Foundation even explicitly says: Do not give your child Aspirin. But even this information did not prompt the study’s authors to investigate the role Aspirin or other substances might have played in the three-year-old’s demise. They spared no trouble, on the other hand, back in 1997 to warn of a rapid and explosive spread of a pandemic virus.

H5N1 fear mongers continue to predict impending horror for Germany. A pandemic will come over us in several waves, Bernhard Ruf, director of the Leipzig Competence Centre for Highly Contagious Diseases and top warrior against avian flu at the WHO, asserts confidently. And we would be lucky to survive the year 2015 without a pandemic. In Germany alone, up to 40 million will become infected and 150,000 will die. The economy will collapse. The world will be paralyzed.

But there are no justifications for such warnings if H5N1 cannot be isolated as a pure virus, and thus cannot scientifically be proven to exist. And if there’s no proof that H5N1 can be highly contagious in animals, by jumping from wild birds to domestic animals and mutating into an infectious mini-monster. And if it cannot be shown that this so-called H5N1 can also jump to humans and cause disease, as a deadly avian flu virus and a human influenza virus come into contact in a human organism, exchange genes, and as evil parent viruses, as they’re called, give birth to an even more horrible daughter virus. And furthermore, if other factors like factory farming, pesticides, rodent poisons, stress and natural death are overlooked as potential contributing factors.

The FLI even admits this to us: Concerning your inquiry about the pandemic properties of H5N1, it can only be said that there are currently no scientific methods with forecasting effects which could evaluate the possibility of an influenza virus triggering a new pandemic. And in late October 2005, the British Medical Journal stated that, the lack of sustained human-to-human transmission suggests that this H5N1 avian virus does not currently have the capacity to cause a human pandemic.

Here it’s worth noting the comments of Julie Gerbering, director of the Centers for Disease Control in Atlanta. In mid-April 2006, at a conference on avian flu pandemic in Tacoma, Washington, with 1200 experts from all over the country in the audience, she said, There is no evidence [H5N1] will be the next pandemic. Further, [there is] no evidence it is evolving in a direction that is becoming more transmissible to people, and there is no reason to think it ever will pass easily between people. These statements are in complete contrast to the continued panic reports by CDC officials. After the conference, The News Tribune reported that, given those facts, bird flu, like SARS, swine flu and other once widely publicized health threats, might never become a significant human illness.

It is scandalous then that, as a result of unfounded pandemic warnings, more than 200 million
birds had been killed by April 2006. Additionally, as a UNO report continued, costs totaling $20 billion had been incurred by the affected countries by this time and a million farmers had already slid into poverty. In Germany, the government ordered that poultry be kept indoors even led to suicide among some breeders. As the Westfalian newspaper Westfalen-Blatt reported the breeders did not see any way out. Indeed, at the very least, ordering small poultry breeders to keep their birds inside is tantamount to banning them from their profession.

There is no foundation for vehement demands for antiviral medications. Nevertheless, mainstream media like Die Zeit insist it is high time that Germany buys vaccines and enough medicine. But just how dangerous are such hasty demands for a quick-fix becomes clear by tracking the rise of Tamiflu, a flu remedy that became a hot-seller only after the virus mania machine cranked up. Tamiflu, conceived as a remedy for common flu, did not sell well because it was too expensive and had too little effect, according to a rare industry critique by the Swiss news magazine Rundschau on 19 October 2005. The pharmaceutical groups promised a lot, but in practice it was shown that doctors could hardly prescribe the medicine to anyone.

So, the virus hunters and their media sidekicks released terrifying pictures of infection experts in white spacesuits and remote factory farms with piles of dead birds. These images were beamed around the globe, accompanied by sensationalized tales of people who had already allegedly become infected with or died from the horrible HSN I virus. In 2004, the WHO office in Manila promptly recommended oseltamivir (Tamiflu) for endangered individuals. The substance was produced by the Swiss pharmaceutical giant Roche, under the brand name Tamiflu.

Roche took advantage of the moment and quickly issued a press release saying, Tamiflu may be effective against avian flu. But the media didn’t seem to take notice of the phrase may be and crafted their headlines to tout a miracle remedy for avian flu. For Roche, this was the best kind of advertising: free and with an incredible effect. Some pharmacies soon sold out of the medication. In the media and television, they always say that Tamiflu works against the avian flu virus, said a pharmacist from Istanbul in an interview with the Rundschau. Now, they all come and want Tamiflu.

Reuters news agency reported on 20 July 2005, that the global flu precautions had granted [Tamiflu manufacturer] Roche a leap in profits. Worldwide, Tamiflu sales increased by 363% to 580 million franks [ € 380 million] in the first half of 2005, in comparison to the same period in the previous year. Ultimately, in 2005, Roche increased its Tamiflu profits by 370% to around € 1 billion90-primarily thanks to massive government purchases (financed by tax dollars). As the Zeit relates, the German province of North Rhine-Westfalia announced that they would put € 30 million worth of medications into storage. 91 In the first nine months of 2006, worldwide Tamiflu sales rose to $1.3 billion, Roche reported , an increase of 88% over the year prior. To keep up with demand, Roche factories in Europe, North America and Japan worked full throttle. By the end of 2006, capacity has doubled once again, to an annual production of 300 million packages of Tamiflu.

But what scientific basis is there for this Tamiflu hype? Franz Humer, Chairman of Roches Board of Directors, assures that Tamiflu is a very important product for our patients, above all in
case of an influenza pandemic. But this statement doesn’t hold up, since Tamiflu has never been
tested as a remedy for avian flu in humans, as even stated by a press release from Roche. In this,
it says that there is no clinical data on the effectiveness of Tamiflu against H5N1.

This is also why Robert Dietz at the World Health Organization in Manila, which jumpstarted
Tamiflu’s sales-explosion with its promotion of the flu remedy, could not avoid admitting to the
Swiss news program Rundschau: We had no specific medical foundation for our decision to
recommend Tamiflu as a remedy for avian flu.

In fact, in early December 2005, the Vietnamese doctor Nguyen Tuong Van, director of the
Intensive Care unit at Hanoi’s Institute for Clinical Research into Tropical Diseases (who had
followed WHO guidelines for patient treatment), came to the conclusion that Tamiflu is useless;
[for this reason,] we place no importance on using this drug on our patients. 95 And just prior to
this statement, appeared the first reports on deaths connected to the intake of Tamiflu.
First came a report from Japan. The pharmaceutical company Chugai, a Roche subsidiary, had
notified the Health Ministry that after Tamiflu intake, two boys aged 14 and 17 became
disoriented, showed abnormal behavior and ultimately died (one was thought to have jumped
from his apartment; the other had thrown himself in front of a truck). Only a few days later, news
made the rounds that the influenza medication was connected to the deaths of twelve children in
Japan. And the American Food and Drug Administration (FDA) called it unsettling that after
Tamiflu intake, children in 32 cases had had hallucinations or shown abnormal behavior.

Of course, these cases are not restricted to Japan. For example, near the end of 2006, Canadian
officials at Health Canada warned of hallucinations among Tamiflu users. As of November 11,
there had been seven cases of psychiatric side effects linked to Tamiflu in Canada and 84 reports
of side effects occurring in Canadians taking the medication, including 10 deaths.

But the media doesn’t push reports of Tamiflu’s side effects nearly as much as the earlier
completely unfounded declarations that Tamiflu was the best protection from avian flu (HSN1).
This is certainly due to the fact that, in connection with the reported fatalities, the medical
establishment immediately warned people not to panic just because a few people had died after
taking Tamiflu—and in the typical manner, the media followed the medical establishment’s
placations. The FDA stressed that they wanted to investigate why people had died, but they
implied that it was extremely difficult to establish the exact causes.

As early as the 1990s, Tamiflu was found to cause inflammations in the brain (encephalitis). But
the medical establishment twisted these findings by saying that neural symptoms were also often
triggered by influenza infections, so they said that it was difficult to tell whether Tamiflu could
be responsible for the neurological complications. This was made even more difficult because
many victims had been taking not just Tamiflu, but also other medications. Basically, the issue
could only be clarified if controlled studies (one group/patient receives the active substance,
the other a placebo) were available. But, they weren’t available.

Why was this medication never tested through the necessary clinical trials before being released
to the public? The information provokes disbelief, particularly since the medical establishment
and the politicians actively participates in virus mania, celebrates medications like Tamiflu and
only calls for caution and restraint when news of medication-related deaths start to circulate. At which point, they rush to the side of the pharmaceutical companies whose bottom lines might be negatively affected.

Just follow the money, as Mark Felt, the FBI's second in command, told Washington Post reporters Bob Woodward and Carl Bernstein during the Watergate scandal in the early 1970s. If it were ever conclusively established that Tamiflu caused deaths, this would be a tragedy of unimaginable scope. It would also be a huge disaster for Roche. But, until clarity prevails, there is no reason to buy or take Tamiflu, neither prophylactically nor as a remedy for flu symptoms. Tamiflu is connected with numerous side effects, including vomiting, diarrhea, bronchitis, stomach · and headaches, dizziness, hallucinations and hepatitis.

A patient who had taken Tamiflu for just two days reports: I couldn’t sleep for three days and I hallucinated. My family was very worried about me. I will never take this horrible medicine again and would not advise anyone to. I completely lost my personality, I felt as if I was a different person. It was four weeks before I started feeling myself again. There must also be studies that show Tamiflu works against flu, right? Of course, such studies would be worthless without placebo controls, along with a guarantee that the scientists involved were free from conflicts of interest. Has the media ever taken the trouble to double check if the Tamiflu trials were sound? We do know one thing for certain: fraud is well established in biomedicine, and conflicts of interest are widespread. Making it urgently necessary to sort fact from fiction.

It doesn’t take much research to find out if Roche has financed Tamiflu (oseltamivir) studies. You only need to Google, for example, Roche funded pubmed oseltamivir—more than 100 hits come up. Let’s click on just one paper: for instance: Effectiveness of neuraminidase inhibitors in treatment and prevention of influenza A and B: systematic review and meta-analyses of randomized controlled trials, published in the British Medical Journal in 2003. It includes the following information: Competing interests: KGN [Karl G. Nicholson; one of the study’s authors] has received travel sponsorship and honorariums from GlaxoSmithKline, the manufacturer of zanamivir, and Roche, which makes oseltamivir, for consultancy and speaking at international respiratory and infectious diseases symposiums. His research group has received research funding from GlaxoSmithKline and Roche to participate in multicenter trials of neuraminidase inhibitors.

Unfortunately, such conflicts of interest are common practice, something to which the public is rarely made aware. But as the British Parliament observed in a comprehensive investigation in 2005, three-quarters of clinical studies that appear in the leading scientific journals, The Lancet, The New England Journal of Medicine (NEJM) and The Journal of the American Medical Association (JAMA), are funded by pharmaceutical companies. And if the industry is paying, they will use all sorts of tricks to attain the desired results, by omitting the critical questions or negative results and exclusively publishing positive results.

Nonetheless, the NEJM explicitly modified its policy for writers in 2002, so that review articles and editorials could also be written by experts who receive fees of up to $10,000 a year from pharmaceutical companies. The fees can also come from companies whose products are plugged by the author in his or her NEJM articles.
This presents a classic conflict of interest. What was the key reason for the alterations to their writer’s policy? The NEJM said that they were simply no longer in a position to find enough experts without any financial connections to the pharmaceutical industry.

For an allegedly independent scientific journal, this explanation seems ludicrous, but it depicts the stark reality of modern medical science. Arnold Reiman, Harvard professor and former Editor in Chief of the NEJM says that, The medical profession is being bought by the pharmaceutical industry, not only in terms of the practice, but also in terms of teaching and research.

Precisely these financial interconnections threaten to undercut the independence of medical research. The issue only recently reached top circles in the USA after it was revealed that hundreds of scientists employed by the National Institutes of Health had received millions of dollars in commissions and big stock packages from the pharmaceutical industry. The story was researched by the Los Angeles Times and triggered a broad discussion on the independence of NIH researchers.

US Congress members accused NIH leaders and their predecessors with supporting the option of corruption among its employees. In response, Elias Zerhouni, the health authority’s director, announced the introduction of new rules which banned higher NIH managers from signing paid consulting contracts, and prohibited all NIH employees from holding stocks and stock options.

But it turned out that many thousand NIH employees were exempt from the obligation to disclose their acquisitions. Through this loophole they could continue to be paid in secret by pharmaceutical companies without fear of punishment. With Tamiflu specifically, doctors and other experts have begun to ask critical questions regarding the US government’s vehement commitment to the purchase of stockpiles of the Roche medication. Death by avian flu, according to President George W. Bush, threatens two million Americans. This statement, based on nothing more than wild speculation, seemed to justify the massive purchase of 20 million bottles of Tamiflu at $100 each. For a total cost of $2 billion.

Particularly alarming is the fact that, at taxpayers’ expense, enormous sums are spent on a medication whose efficacy against avian flu has never been proven and will never be proven either. For, even assuming that HSN1 does exist and causes disease in humans, nobody can predict what the mutated form of the HSN1 virus, which is supposed to first trigger the pandemic, will look like. This means that no medication, not even Tamiflu, can be conceived against such an alleged mutant virus.

And this is exactly why the UK government’s decision to order 14.6 million doses of oseltamivir for use in the event of a flu epidemic has been questioned even by orthodox experts. Among them Joe Collier, professor of medicines policy at St Georges Hospital Medical School, London, and former editor of the Drug and Therapeutics Bulletin who has been quoted in the British Medical Journal with the words: I would like to know what evidence there is that Tamiflu actually alters mortality. And if it doesn’t then what are we doing?
On the other side of the Atlantic Canada’s federal health minister, Ujjal Dosanjh, told listeners to an interview on a Canadian Broadcasting Corporation radio program (The Current, 27 October 2005) that oseltamivir did not prevent infection with the flu virus.

This is why it many were upset that Donald Rumsfeld, a leading member of the George W. Bush administration, was making money thanks to massive state Tamiflu purchases. As a once-leading member of the Bush administration, he makes a tidy sum of cash from massive state Tamiflu purchases. From 1997 until 2001, before taking office, Rumsfeld chaired the Board of Directors of the American biotechnology corporation Gilead. And after 2001, according to his own statements, Rumsfeld continued to hold huge share packages in Gilead valued at $5 - 25 million. Gilead had originally developed Tamiflu, and in 1997, the Nasdaq-listed corporation sold an exclusive license to Roche for the production of Tamiflu, though Gilead kept the substances patent.

Gilead has since cashed in license fees from Roche (as is reported, between 10% and 19% of net price, or 10% of profits). In the three (hot) autumn months of 2005, Tamiflu licensing brought in $12 million for Gilead; up from $1.7 million in the third quarter of 2004. Simultaneously, Gilead market values climbed from $37 to $47 within just a few months, something that made Rumsfeld-one of the richest men in the Bush cabinet-at least $ 1 million richer.

Rumsfeld isn’t the only political heavyweight in the USA, who is said to have very close connections to Gilead. George P. Shultz, US Secretary of State from 1982 to 1989, is on Gilead’s Board of Directors. In 2005, Shultz sold stocks of the Californian biotech company at a value of more than $7 million. Another member of Gilead’s board is the wife of former California governor Pete Wilson. I don’t know of any biotech company that’s so politically well-connected [as Gilead] , Andrew McDonald, of the analyst firm Think Equity Partners, told Fortune. A Saar-Echo article, published under the title Bush Makes Panic and Rumsfeld Profit, hits the nail on the head: Bush and his vice-president, Dick Cheney, the human embodiment of the combination of oil and military interests had developed the pattern of this capitalistic escapade for the good of the American billionaires oligarchy in connection with the Iraq War, when they explained their invasion of the oil-rich Middle Eastern country with the shameless lie that Iraq was in possession of weapons of mass destruction.

After the defeat of Sad dam Hussein, one of the main profiteers from the Iraq invasion was the American company Halliburton, whose core business is trade and conveyance of crude oil. The CEO of Halliburton, until his leap to the seat of the American vice-president, was Richard Cheney, who in turn is a close friend of Tamiflu profiteer Donald Rumsfeld. Together, they founded the neoconservative think tank Project for the New American Century in 1997. Since they have held office, the billion-dollar side projects of these and other US politicians have run like clockwork.

Although massive accusations of fraud are levied against Halliburton, because, for example, the group charges exorbitant prices for many services (for the cleaning of just 7 kilograms of laundry, more than $ 100 was charged) , the US Army placed a new order in 2005 to support the
troops in Iraq. The price tag: $5 billion. In 2004 and 2003, the oil and gas subcontractor based in Texas, George W. Bush’s home state, had already pocketed $10 billion.

In his farewell speech in 1961, outgoing president Dwight D. Eisenhower warned of the increasing entanglement of military and industry, and of the growing influence of this military-industrial complex on American politics. This enlightened warning was repeated in the award-winning documentary Why We Fight, a focus on today’s billion-dollar war machine. 40 years later, history seems to be proving Eisenhower right.

One of the many parallels between the military-industrial complex and the medical-industrial complex is huge funding by tax dollars. In 2005, the Bush administration announced that they were introducing a $7.1 billion program to protect the USA from a possible avian flu epidemic. Just a few weeks before, Bush had been heavily criticized around crisis management in New Orleans after Hurricane Katrina. Ironic as it may seem, the government saw an excellent opportunity to polish up Bush’s battered public image in the announcement of an (incredibly expensive taxpayer funded) avian flu package.

According to Bush, they wanted to buy enough vaccine against the avian virus to protect 20 million Americans. For this, they would attempt to get the US Congress to approve $1.2 billion. Additionally, they hoped to get approval of nearly $3 billion for the development of new flu vaccines, as well as $1 billion for the storage of antiviral medications. And a further $600 million was allocated for local authorities, so that they could create emergency plans for containment of an epidemic. Bush also demanded that Congress ease liability regulations for vaccine manufacturers. Only this way, it was said, could production capacity grow, since pharmaceutical firms refused to manufacture vaccines without protection from damage lawsuits. Of course, from a consumer perspective, if such a scheme were to become reality, Americans who suffered vaccine-related damages would be denied the basic right to claim damage or other compensation by way of the law.

This plan is part of a legal initiative—the Biodefense and Pandemic Vaccine and Drug Development Act of 2005—which would allow no more lawsuits, even if vaccinations or medications are administered by force. A drug company stockholders dream and a consumer’s worst nightmare, according to the National Vaccine Information Center.

Not to be swayed by scientific interest groups, Bush countered back with, No country can afford to ignore the threat of avian flu. He did admit that nobody knew if the HSN1 flu virus could lead to a deadly human epidemic, but he warned that history dictates we must once again anticipate a terrible large epidemic. Bush was referring to the so-called Spanish flu of 1918, to which many millions of people fell victim. This Spanish flu was so named because the Spanish media were the only ones to report about the virus while most other nations decreed an information ban on the pandemic, allegedly in order to avoid fear among World War I troops. But is it really a suitable virus model for any sort of pandemic predictions nowadays?

Within a few months, the Spanish flu achieved what all the epidemics in history have not managed, wrote Spiegel Online. In 1918, the pandemic killed between 20 and 50 million people, more than any other disease before. In the USA alone, there were 550,000 deaths. Infected
patients suffered from high fever and their lungs became inflamed. Within a few days, victims drowned in their own fluids.

It sounds dramatic-and it was dramatic. But it’s much too hasty to assume that a virus triggered mass mortality. There are certainly no facts to support such a theory. These mass deaths occurred at the end of the First World War (July 1914 to November 1918), at a time when countless people were undernourished and under incredible stress after four years of war.

Additionally, the medications and vaccines applied in masses at that time contained highly toxic substances like heavy metals, arsenic, formaldehyde and chloroform, all of which could very likely trigger severe flu symptoms. Numerous chemicals intended for military use also moved unregulated into the public sector (agriculture, medicine).

In 1997, a paper by Jeffery Taubenbergers research team appeared in Science, claiming to have isolated an influenza virus (H 1N1) from a victim of the 1918 pandemic. But before one can be certain that a pandemic virus had in fact been detected, some important questions must be asked, writes Canadian biologist David Crowe, who analyzed the paper.

The researchers had taken genetic material from the preserved lung tissue of a victim—a soldier, who died in 1918. Lung diseases were extremely typical of the Spanish flu, but it is a big leap to conclude that the many other million victims also died from the same cause. And particularly the same virus as Crowe points out.

We simply do not know if the majority of victims died for exactly the same reason. We also do not know if a virus can be held responsible for all mortalities, because viruses, as they’re now be described, were unknown at this time. Even if one does accept that an influenza virus was present in the soldier’s lungs, this hardly means that this virus was the killer.

Taubenbergers group admits that the soldier was an atypical case, since most of the so-called influenza victims (influenza suggests a viral cause) actually died from bacterial lung inflammations (for example, tuberculosis). These bacteria, it is conjectured, ultimately gained the upper hand and supplanted the viruses. But this speculation doesn’t necessarily make any sense. The genetic analysis of pulmonary tissue form the single soldier was based on the assumption that certain genetic sequences (RNA sequences) are characteristic of all flu viruses. That is, it is theorized that there are certain proteins in flu virus shells, the RNA sequences of which were ultimately claimed to have been discovered using PCR. These proteins are hemagglutinins (this is where the H in H1N1 or HSN1 comes from: H1 and HS stand for certain hemagglutinin types) and neuraminidases (the N). But in biochemistry, many different substances are termed hemagglutinins, not just proteins that cause red blood cells to clot together. Nevertheless, it is said that proof of a virus can be exhibited by mixing red blood cells in the laboratory with samples, in which the alleged virus is said to be found. This was done by taking tissue samples from organs in which the virus is presumed to lurk (in this case from a lung) in placing them (in vitro) into a petri dish filled with red blood cells. If clots then form, the theory goes that a hemagglutinins in a flu virus must have been the cause of the coagulation.

But a complete virus had never been isolated from this sample. This method is also weak since it cannot differentiate between the RNA of an external virus and human RNA. This cannot be
normal human RNA, otherwise everyone would react positively to the method, says Crowe. But it would certainly be possible that the RNA collected by the PCR does not come from a virus protein, but is rather produced by the body itself, for instance in connection with a disease process.

The enzyme neuraminidase, for instance, which is held to be specific to a flu virus, is actually produced naturally by the body and performs significant metabolic functions. If there is a deficiency of this enzyme—because of an innate metabolism disorder, for example—orthodox medicine has long called this Mucolipidosis or Sialidosis which causes serious dysfunctions such as impaired vision, disorders of the nervous system and the skeleton, myasthenia (muscle weakness), seizures, disturbances of equilibrium, or cerebral development disorders. Anyone who takes flu remedies and neuraminidase inhibitors like Tamiflu should keep this in mind. We can then conclude that Taubenberger et al, have not verifiably shown that a flu virus was present in the soldier. Their experiment cannot prove that this soldier died from a flu virus, let alone that the other umpteen million victims lost their lives because of a specific virus.

The same is true of the papers published in the scientific journals Nature and Science in October 2005. The media reports spun the information into a global sensation with news that US researchers revive old killer virus and American scientists have reconstructed the extremely dangerous Spanish flu pathogen in a military laboratory. But even if headlines suggest this, the fact is that here as well, a virus with complete genetic material (genome) had never been discovered. Lung tissue samples were simply taken from several corpses from that time, including an Inuit woman buried in Alaska’s permafrost layer in 1918. Then, the scientists conducted practically the same procedure as in 1997. Researchers had not proven that the genetic material they found really belongs to a pathogenic old killer virus. With many samples, the tests even came out negative. The whole thing, then, is pure speculation.

According to traditional conceptions, an infectious disease begins in one place and spreads out from there, depending on the environmental conditions, in certain directions. Such a development didn’t occur with the Spanish flu.

In 1918, there were two different disease waves: a lighter one in spring and a much more severe wave, which claimed many lives, in late summer and autumn. Here, experts can’t even agree whether the disease was introduced to the United States from Europe, or the other way around. According to one source, the epidemic began in February 1918 in the Spanish town of San Sebastian, close to the French border on the Atlantic coast. But another source names the same outbreak date, but a completely different place thousands of kilometers away from San Sebastian, on the other side of the Atlantic: New York City. That these outbreaks happened at the same time cannot be explained by either ship route or migrating bird patterns.

Then in March 1918, there were reports of cases in two army camps in Kansas, hundreds of kilometers away from New York. In April, the Spanish flu appeared in Paris for the first time, in May in Madrid, until it reached its peak in Spain at the end of May. In June, cases first began accumulating in war-tom Germany, but simultaneously in China, Japan, England and Norway as well. On 1 July, Leipzig had its first case. And over the course of that month, approximately half a million Germans were affected.
The second serious wave began almost at the same time in Boston’s Harbor, on the Indian subcontinent, in Southeast Asia, in the Caribbean and Central America. In September, various army camps in the western USA along with the states of Massachusetts, Pennsylvania and Philadelphia were affected. In October Brazil was hit, and in November Alaska.

But even if we factor in the fastest ships of the time, railway routes and migrating birds, there’s no sound epidemiological basis to construct a virus-caused influenza. Unless one assumes that the virus mutated into a deadly infectious agent on all continents simultaneously—which is probably less likely than winning the lottery ten times in a row.

In order to be able to better assess the puzzling mass disease, an attempt to simulate infection was undertaken with volunteers in Boston in November 1918. These were 62 healthy sailors charged with delinquency and sent to prison. They had been promised a pardon under the condition that they take part in an experiment.

39 of them had not had influenza, so the theory was that they would be particularly susceptible to infection and illness. But the results proved nothing of the sort, as American scientific journalist Gina Kolata describes in her book Influenza: Navy doctors collected the mucus from men who were desperately ill from the flu, gathering thick viscous secretions from their noses and throats. They sprayed mucus from flu patients into the noses and throats of some men and dropped it into other men’s eyes. In one attempt, they swabbed mucus from the back of the nose of a man with the flu and then directly swabbed one patient’s nasal septum and rubbed it directly onto the nasal septum of one of the volunteers.

Trying to simulate what happens naturally when people are exposed to flu victims, the doctors took ten of the volunteers onto the hospital ward where men were dying of the disease. The sick men lay huddled on their narrow beds, burning with fever, drifting in and out of sleep in a delirium. The ten healthy men were given their instructions: each was to walk up to the bed of a sick man and draw near him, lean into his face, breathe in his fetid breath, and chat with him for five minutes. To be sure that the healthy man had had a full exposure to the sick man’s disease, the sick man was to exhale deeply while the healthy man drew the sick man’s breath directly into his own lungs. Finally, the flu victim coughed five times in the volunteers face.

Each healthy volunteer repeated these actions with ten different flu patients. Each flu patient had been seriously ill for no more than three days—a period when the virus or whatever it was that was causing the flu should still be around in his mucus, in his nose, in his lungs. But not a single healthy man got sick.

A comparable experiment, carried out under much stricter conditions, took place in San Francisco, with 50 imprisoned sailors. But, once again, the results did not correspond with what the doctors had expected: Scientists were stunned. If these healthy volunteers did not get infected with influenza despite doctor’s best efforts to make them ill, then what was causing this disease? How, exactly, did people get the flu?

A look at history books and statistics shows that epidemics always developed where human
immune systems had been weakened, primarily because of lack of food and clean water. This was also the case with the pandemic of 1918. A panoply of causes, which naturally could also have worked in combination, comes into consideration:
- Psychological stress, evoked by fears of war
- Over-treatment with chemical preparations, which can seriously compromise the immune system, including painkillers like Aspirin or chloroform. Chloroform, which was used as a preservative in medications, and transformed into phosgene in the body [liver], which was used as poison gas in the First World War. In the late 19th century, manufacturers of medicinal products also increasingly began selling products that contained highly toxic substances like morphine, codeine, quinine and strychnine as medicines; at that time there were no regulations for such manufacturers. From 1898, the German inventor of Aspirin, Bayer, sold heroin, for example, as an allegedly non-addictive morphine substitute, and also as a cough remedy in many different forms, ranging from syrup-in noble-looking flacons-to plugs, powders, liquids, and tampons soaked in it for gynecological treatments.
- Damage to airway organs resulting from preventive measures, like rubbing the throat with antiseptic preparations or inhaling antibacterial substances. Many of the substances used at that time also contained silver and have long been prohibited (for example, Formalin/formaldehyde has strong corrosive and irritating effects on skin, eyes, and airway, and can cause kidney, liver and lung damage; a carcinogenic potential is also attributed to it)
- No effective antibiotics: many people’s were afflicted by bacterial and fungal infections, but the first really effective means of killing bacteria and fungi was penicillin, which was discovered much later, in 1928, and became a medication during the Second World War
- Vaccines often contained toxic heavy metals and were produced out of poorly filtered mucus or other fluids from infected patients

A frequently observed symptom of the Spanish flu was internal bleeding in the lungs (typical of tuberculosis patients, for example)—a phenomenon that was also described as a result of smallpox vaccinations. In fact, numerous sources report that mass vaccinations (up to 24 vaccinations per person) decisively contributed to the pandemic. American author Eleanora McBean relates her own experiences: All the doctors and people who were living at the time of the 1918 Spanish Influenza epidemic say it was the most terrible disease the world has ever had. Strong men, hale and hearty, one day would be dead the next. The disease had the characteristics of the Black Death added to typhus, diphtheria, pneumonia, smallpox, paralysis and all the diseases the people had been vaccinated with immediately following World War 1. Practically the entire population had been injected/seeded with a dozen or more diseases—or toxic serums. When all those doctor-made diseases started breaking out all at once it was tragic.

That pandemic dragged on for two years, kept alive with the addition of more poison drugs administered by the doctors who tried to suppress the symptoms. As far as I could find out, the flu hit only the vaccinated. Those who had refused the shots escaped the flu. My family had refused all the vaccinations so we remained well all the time. We knew from the health teachings of Graham, Trail, Tilden and others, that people cannot contaminate the body with poisons without causing disease.

When the flu was at its peak, all the stores were closed as well as the schools, businesses—even
the hospital, as the doctors and nurses had been vaccinated too and were down with the flu. No one was on the streets. It was like a ghost town. We seemed to be the only family [that] didn’t get the flu; so my parents went from house to house doing what they could to look after the sick, as it was impossible to get a doctor then. If it were possible for germs, bacteria, virus, or bacilli to cause disease, they had plenty of opportunity to attack my parents when they were spending many hours a day in the sick rooms. But they didn’t get the flu and they didn’t bring any germs home to attack us children and cause anything. None of our family had the flu—not even a sniffle—and it was in the winter with deep snow on the ground.

When I see people cringe when someone near them sneezes or coughs, I wonder how long it will take them to find out that they can’t catch it—whatever it is. The only way they can get a disease is to develop it themselves by wrong eating, drinking, smoking or doing some other things which cause internal poisoning and lowered vitality. All diseases are preventable and most of them are curable with the right methods, not known to medical doctors, and not all drugless doctors know them either.

It has been said that the 1918 flu epidemic killed 20 million people throughout the world. But, actually, the doctors killed them with their crude and deadly treatments and drugs. This is a harsh accusation but it is nevertheless true, judging by the success of the drugless doctors in comparison with that of the medical doctors.

While the medical men and medical hospitals were losing 33% of their flu cases, the non-medical hospitals such as Battle Creek, Kellogg and McFadden’s Health Restorium were getting almost 100% healings with their water cure, baths, enemas, etc., fasting and certain other simple healing methods, followed by carefully worked out diets of natural foods. One health doctor didn’t lose a patient in eight years.

If the medical doctors had been as advanced as the drugless doctors, there would not have been those 20 million deaths from the medical flu treatment. There was seven times more disease among the vaccinated soldiers than among the unvaccinated civilians, and the diseases were those they had been vaccinated against. One soldier who had returned from overseas in 1912 told me that the army hospitals were filled with cases of infantile paralysis [polio] and he wondered why grown men should have an infant disease. Now, we know that paralysis is a common after-effect of vaccine poisoning. Those at home didn’t get the paralysis until after the world-wide vaccination campaign in 1918.

Author Anne Riley Hale alludes to all of the above factors in her 1935 book Medical Voodoo: As everyone knows, the world has never witnessed such an orgy of vaccination and inoculation of every description as was inflicted by army-camp doctors upon the soldiers of the [First] World War. Hale also observed that the amazing disease and death toll among them occurred among the picked men of the nation—supposedly the most robust, resistant class of all, who presumably brought to the service each a good pair of lungs, since they must have passed a rigid physical examination by competent medical men. And yet, precisely these supermen with super-lungs were the ones who were dropping like flies from pulmonary tuberculosis.

In this context, a report in the Idaho Observer (July 2003) is also worth noting. It mentions a
contemporary vaccination trial by one Dr. Rose now, published in the Mayo Collected Papers of the world-renowned Mayo Clinic. According to this paper, the vaccinated guinea pigs primarily suffered severe damage in their lungs—a typical symptom of tuberculosis and other diseases of the Spanish flu.

Meanwhile, medical historians are amazed that doctors and the media have remained silent about the catastrophes that resulted from Spanish flu. As Kalata writes in her book, Victor Vaughan, at that time, America's top military doctor, dealt with the mega-catastrophe in just one paragraph of his 464 page long memoirs. And yet, Vaughan must have recollected everything very well, as his book appeared in 1926, not long after the war's end (and he probably would never forget the horrific events). If anyone might be expected to write about the epidemic it was Vaughan, writes Kalata. Like Vaughn, other army doctors remained steadfastly silent.

The pandemic, one of the worst to ever afflict the earth, was simply virtually erased from newspapers, magazines, books and society’s collective memory, says Kolata. This could be psychologically explained in two ways. The catastrophe presented a very personal catastrophe for physicians, because, although they were basically given all the money and material resources in their world to fight the alleged flu, they were unsuccessful in preventing the disaster. In a brutally clear way, doctors and pharmacologists were shown the limits of their power. It is clear that mainstream medicine prefers not to dwell on such a total defeat, let alone expand upon it in memoirs or newspapers.

Perhaps the occasional scientist, doctor or politician began to mull over the lost campaign against an imaginary virus and entertained the thought that the mass administration of highly toxic vaccines and medications could have been at least partially responsible for the pandemic. Clues for this were by all means visible. But who likes to take responsibility for the deaths of millions of people—even unintentionally—and admit failure to fulfill the duty to investigate all factors that come into question?

Chapter 8

Louis Pasteur, Robert Koch and their heirs have inoculated us with a monocausal theory of disease. The picture is alluring and comforting because it completely shifts the blame away from ourselves to microbes, and suggests that if we simply throw enough money at pharmaceutical research—presto!—we were safe from all sorts of diseases, including flu. But we’re still waiting for side effect-free miracle pills that will liberate us from flu symptoms.

Mainstream medicine holds that flu medications and vaccines have worked wonders. But a glance in history books and statistics reveals, as mentioned, proves that these so-called epidemics only developed when peoples immune systems had been weakened, starting with lack of food or clean water and compounded by chemical toxins like medications, warfare agents and pesticides. The diseases, held to be caused primarily by viruses, had long begun their retreat when vaccine campaigns were finally introduced. For example, population statistics in the USA show that the death rates in senior citizens were quite stable from 1980 onwards, although the vaccination rate had climbed steeply from 1980 to 2001 (from 15 to 65%)—and parallel to this, the number of flu victims had also climbed.
Most people probably think vaccinations are sensible. And typically most critics of vaccinations believe that today’s vaccines contain fewer toxins than they did in the past. But ultimately, nobody knows what is really in the substances and it difficult to gather information about them. Even today, they are certainly not safe, says vaccine expert Angelika Kogei-Schauz. Studies have shown that vaccines trigger serious cases of Guillain-Barre syndrome, a disease that is associated with polio-like neural damage.

Many vaccine serums still contain thimerosal, a preservative which is made up of up to 50% mercury. Thimerosal is strongly suspected of triggering autism, according to a comprehensive 2003 report. In 2005, this subject was heatedly debated in the USA, even by major media, after journalist David Kirby had collected the data relevant to this issue and published it in his book Evidence of Harm. Mercury in Vaccines and the Autism Epidemic-A Medical Controversy. Grounded suspicion now exists that many factors, such as pesticides or organic toxins like PCB-and particularly the mercury contained in vaccines-are connected with autism cases, the rate of which has expanded to sixty times its size since 1980.

Robert F. Kennedy Jr. investigates the government cover-up of a mercury/autism scandal (Originally published June 2005 by Rolling Stone magazine and Salon.com, updated in 2006). In June 2000, a group of top government scientists and health officials gathered for a meeting at the isolated Simpsonwood conference center in Norcross, Georgia. Convened by the Centers for Disease Control and Prevention, the meeting was held at this Methodist retreat center, nestled in wooded farmland next to the Chattahoochee River, to ensure complete secrecy. The agency had issued no public announcement of the session-only private invitations to 52 attendees.

There were high-level officials from the CDC and the Food and Drug Administration, the top vaccine specialist from the World Health Organization in Geneva and representatives of every major vaccine manufacturer, including GlaxoSmithKline, Merck, Wyeth and Aventis Pasteur. All of the scientific data under discussion, CDC officials repeatedly reminded the participants, was strictly embargoed. There would be no making photocopies of documents, no taking papers with them when they left.

The federal officials and industry representatives had assembled to discuss a disturbing new study that raised alarming questions about the safety of a host of common childhood vaccines administered to infants and young children. According to a CDC epidemiologist named Tom Verstraeten, who had analyzed the agency’s massive database containing the medical records of 100,000 children, a mercury-based preservative in the vaccines-thimerosal—appeared to be responsible for a dramatic increase in autism and a host of other neurological disorders among children.

I was actually stunned by what I saw, Verstraeten told those assembled at Simpsonwood, citing the staggering number of earlier studies that indicate a link between thimerosal and speech delays, attention-deficit disorder, hyperactivity and autism. Since 1991, when the CDC and the FDA had recommended that three additional vaccines laced with the preservative be given to extremely young infants— in one case, within hours of birth—the estimated number of cases of autism had increased fifteen fold, from one in every 2,500 children to one in 166 children.
Even for scientists and doctors accustomed to confronting issues of life and death, the findings were frightening. You can play with this all you want, Dr. Bill Weil, a consultant for the American Academy of Pediatrics, told the group. The results are statistically significant. Dr. Richard Johnston, an immunologist and pediatrician from the University of Colorado whose grandson had been born early on the morning of the meetings first day, was even more alarmed. My gut feeling? he said. Forgive this personal comment-! do not want my grandson to get a thimerosal-containing vaccine until we know better what is going on.

But instead of taking immediate steps to alert the public and rid the vaccine supply of thimerosal, the officials and executives at Simpsonwood spent most of the next two days discussing how to cover up the damaging data. According to transcripts obtained under the Freedom of Information Act, many at the meeting were concerned about how the damaging revelations about thimerosal would affect the vaccine industry’s bottom line. We are in a bad position from the standpoint of defending any lawsuits, said Dr. Robert Brent, a pediatrician at the Alfred I. DuPont Hospital for Children in Delaware. This will be a resource to our very busy plaintiff attorneys in this country. Dr. Bob Chen, head of vaccine safety for the CDC, expressed relief that given the sensitivity of the information, we have been able to keep it out of the hands of, let’s say, less responsible hands. Dr. John Clements, vaccines advisor at the World Health Organization, declared that perhaps this study should not have been done at all.

He added that the research results have to be handled, warning that the study will be taken by others and will be used in other ways beyond the control of this group. In fact, the government has proved to be far more adept at handling the damage than at protecting children’s health. The CDC paid the Institute of Medicine to conduct a new study to whitewash the risks of thimerosal, ordering researchers to rule out the chemicals link to autism. It withheld Verstraeten’s findings, even though they had been slated for immediate publication, and told other scientists that his original data had been lost and could not be replicated.

And to thwart the Freedom of Information Act, it handed its giant database of vaccine records over to a private company, declaring it off-limits to researchers. By the time Verstraeten finally published his study in 2003, he had gone to work for GlaxoSmithKline and reworked his data to bury the link between thimerosal and autism.

Vaccine manufacturers had already begun to phase thimerosal out of injections given to American infants—but they continued to sell off their mercury-based supplies of vaccines until last year. The CDC and FDA gave them a hand, buying up the tainted vaccines for export to developing countries and allowing drug companies to continue using the preservative in some American vaccines—including several pediatric flu shots as well as tetanus boosters routinely given to eleven-year-olds.

The drug companies are also getting help from powerful lawmakers in Washington. Senate Majority Leader Bill Prist, who has received $873,000 in contributions from the pharmaceutical industry, has been working to immunize vaccine makers from liability in 4,200 lawsuits that have been filed by the parents of injured children. On five separate occasions, Prist has tried to seal all of the government’s vaccine-related documents—including the Simpsonwood transcripts—and shield Eli Lilly, the developer of thimerosal, from subpoenas.
In 2002, the day after Prist quietly slipped a rider known as the Eli Lilly Protection Act into a homeland security bill, the company contributed $10,000 to his campaign and bought 5,000 copies of his book on bioterrorism. The measure was repealed by Congress in 2003—but earlier this year, Prist slipped another provision into an antiterrorism bill that would deny compensation to children suffering from vaccine related brain disorders. The lawsuits are of such magnitude that they could put vaccine producers out of business and limit our capacity to deal with a biological attack by terrorists, says Dean Rosen, health policy adviser to Prist.

Even many conservatives are shocked by the government’s effort to cover up the dangers of thimerosal. Rep. Dan Burton, a Republican from Indiana, oversaw a three-year investigation of thimerosal after his grandson was diagnosed with autism. Thimerosal used as a preservative in vaccines is directly related to the autism epidemic, his House Government Reform Committee concluded in its final report. This epidemic in all probability may have been prevented or curtailed had the FDA not been asleep at the switch regarding a lack of safety data regarding injected thimerosal, a known neurotoxin. The FDA and other public-health agencies failed to act, the committee added, out of institutional malfeasance for self protection and misplaced protectionism of the pharmaceutical industry.

The story of how government health agencies colluded with Big Pharma to hide the risks of thimerosal from the public is a chilling case study of institutional arrogance, power and greed. I was drawn into the controversy only reluctantly. As an attorney and environmentalist who has spent years working on issues of mercury toxicity, I frequently met mothers of autistic children who were absolutely convinced that their kids had been injured by vaccines. Privately, I was skeptical.

I doubted that autism could be blamed on a single source. I tended to agree with skeptics like Rep. Henry Waxman, a Democrat from California, who criticized his colleagues on the House Government Reform Committee for leaping to conclusions about autism and vaccinations. Why should we scare people about immunization, Waxman pointed out at one hearing, until we know the facts?

It was only after reading the Simpsonwood transcripts, studying the leading scientific research and talking with many of the nation’s pre-eminent authorities on mercury that I became convinced that the link between thimerosal and the epidemic of childhood neurological disorders is real. Five of my own children are members of the Thimerosal Generation-those born between 1989 and 2003-who received heavy doses of mercury from vaccines.

The elementary grades are overwhelmed with children who have symptoms of neurological or immune-system damage, Patti White, a school nurse, told the House Government Reform Committee in 1999. Vaccines are supposed to be making us healthier; however, in twenty-five years of nursing I have never seen so many damaged, sick kids. Something very, very wrong is happening to our children.

More than 500,000 American kids currently suffer from autism, and pediatricians diagnose more than 40,000 new cases every year. The disease was unknown until 1943, when it was identified
and diagnosed among eleven children born in the months after thimerosal was first added to baby vaccines in 1931.

Some skeptics dispute that the rise in autism is caused by thimerosal-tainted vaccinations. They argue that the increase is a result of better diagnosis—a theory that seems questionable at best, given that most of the new cases of autism are clustered within a single generation of children. If the epidemic is truly an artifact of poor diagnosis, scoffs Dr. Boyd Haley, one of the world’s authorities on mercury toxicity, then where are all the twenty-year-old autistics? Other researchers point out that Americans are exposed to a greater cumulative load of mercury than ever before, from contaminated fish to dental fillings, and suggest that thimerosal in vaccines may be only part of a much larger problem. It’s a concern that certainly deserves far more attention than it has received—but it overlooks the fact that the mercury concentrations in vaccines dwarf other sources of exposure to our children.

What is most striking is the lengths to which many of the leading detectives have gone to ignore—and cover up—the evidence against thimerosal. From the very beginning, the scientific case against the mercury additive has been overwhelming. The preservative, which is used to stem fungi and bacterial growth in vaccines, contains ethylmercury, a potent neurotoxin. Truckloads of studies have shown that mercury tends to accumulate in the brains of primates and other animals after they are injected with vaccines—and that the developing brains of infants are particularly susceptible.

In 1977, a Russian study found that adults exposed to much lower concentrations of ethylmercury than those given to American children still suffered brain damage years later. Russia banned thimerosal from children’s vaccines twenty years ago, and Denmark, Austria, Japan, Great Britain and all the Scandinavian countries have since followed suit.

You couldn’t even construct a study that shows thimerosal is safe, says Haley, who heads the chemistry department at the University of Kentucky. It’s just too dam toxic. If you inject thimerosal into an animal, its brain will sicken. If you apply it to living tissue, the cells die. If you put it in a petri dish, the culture dies. Knowing these things, it would be shocking if one could inject it into an infant without causing damage.

Internal documents reveal that Eli Lilly, which first developed thimerosal, knew from the start that its product could cause damage—and even death—in both animals and humans. In 1930, the company tested thimerosal by administering it to twenty-two patients with terminal meningitis, all of whom died within weeks of being injected—a fact Lilly didn’t bother to report in its study declaring thimerosal safe. In 1935, researchers at another vaccine manufacturer, Pittman-Moore, warned Lilly that its claims about thimerosal’s safety did not check with ours. Half the dogs Pittman injected with thimerosal-based vaccines became sick, leading researchers there to declare the preservative unsatisfactory as a serum intended for use on dogs.

In the decades that followed, the evidence against thimerosal continued to mount. During the Second World War, when the Department of Defense used the preservative in vaccines on soldiers, it required Lilly to label it poison. In 1967, a study in Applied Microbiology found that thimerosal killed mice when added to injected vaccines. Four years later, Lilly’s own studies
discerned that thimerosal was toxic to tissue cells in concentrations as low as one part per million-100 times weaker than the concentration in a typical vaccine. Even so, the company continued to promote thimerosal as nontoxic and also incorporated it into topical disinfectants. In 1977, ten babies at a Toronto hospital died when an antiseptic preserved with thimerosal was dabbed onto their umbilical cords.

In 1982, the FDA proposed a ban on over-the-counter products that contained thimerosal, and in 1991 the agency considered banning it from animal vaccines. But tragically, that same year, the CDC recommended that infants be injected with a series of mercury-laced vaccines. Newborns would be vaccinated for hepatitis B within twenty-four hours of birth, and two-month-old infants would be immunized for Haemophilus influenza B and diphtheria-tetanus-pertussis.

The drug industry knew the additional vaccines posed a danger. The same year that the CDC approved the new vaccines, Dr. Maurice Hilleman, one of the fathers of Merck's vaccine programs, warned the company that six-month-olds who were administered the shots would suffer dangerous exposure to mercury. He recommended that thimerosal be discontinued, especially when used on infants and children, noting that the industry knew of nontoxic alternatives. The best way to go, he added, is to switch to dispensing the actual vaccines without adding preservatives.

For Merck and other drug companies, however, the obstacle was money. Thimerosal enables the pharmaceutical industry to package vaccines in vials that contain multiple doses, which require additional protection because they are more easily contaminated by multiple needle entries. The larger vials cost half as much to produce as smaller, single-dose vials, making it cheaper for international agencies to distribute them to impoverished regions at risk of epidemics. Faced with this cost consideration, Merck ignored Hilleman's warnings, and government officials continued to push more and more thimerosal-based vaccines for children.

Before 1989, American preschoolers received eleven vaccinations-for polio, diphtheria-tetanus-pertussis and measles-mumps-rubella. A decade later, thanks to federal recommendations, children were receiving a total of twenty-two immunizations by the time they reached first grade. As the number of vaccines increased, the rate of autism among children exploded. During the 1990s, 40 million children were injected with thimerosal-based vaccines, receiving unprecedented levels of mercury during a period critical for brain development. Despite the well-documented dangers of thimerosal, it appears that no one bothered to add up the cumulative dose of mercury that children would receive from the mandated vaccines. What took the FDA so long to do the calculations? Peter Patriarca, director of viral products for the agency, asked in an e-mail to the CDC in 1999. Why didn’t CDC and the advisory bodies do these calculations when they rapidly expanded the childhood immunization schedule?

But by that time, the damage was done. At two months, when the infant brain is still at a critical stage of development, infants routinely received three inoculations that contained a total of 62.5 micrograms (μg) of ethylmercury—a level 99 times greater than the EPA's (Environmental Protection Agency) limit for daily exposure to methylmercury, a related neurotoxin. Although the vaccine industry insists that ethylmercury poses little danger because it breaks down rapidly and is removed by the body, several studies—including one published in April by the National
Institutes of Health suggest that ethylmercury is actually more toxic to developing brains and stays in the brain longer than methylmercury.

Officials responsible for childhood immunizations insist that the additional vaccines were necessary to protect infants from disease and that thimerosal is still essential in developing nations, which, they often claim, cannot afford the singledose vials that don’t require a preservative. Dr. Paul Offit, one of CDC's top vaccine advisers, told me, I think if we really have an influenza pandemic—and certainly we will in the next twenty years, because we always do—there’s no way on God's earth that we immunize 280 million people with single-dose vials. There has to be multidose vials.

But while public-health officials may have been well-intentioned, many of those on the CDC advisory committee who backed the additional vaccines had close ties to the industry. Dr. Sam Katz, the committee’s chair, was a paid consultant for most of the major vaccine makers and was part of a team that developed the measles vaccine and brought it to licensure in 1963. Dr. Neal Halsey, another committee member, worked as a researcher for the vaccine companies and received honoraria from Abbott Laboratories for his research on the hepatitis B vaccine.

Indeed, in the tight circle of scientists who work on vaccines, such conflicts of interest are common. Rep. Burton says that the CDC routinely allows scientists with blatant conflicts of interest to serve on intellectual advisory committees that make recommendations on new vaccines, even though they have interests in the products and companies for which they are supposed to be providing unbiased oversight.

The House Government Reform Committee discovered that four of the eight CDC advisers who approved guidelines for a rotavirus vaccine had financial ties to the pharmaceutical companies that were developing different versions of the vaccine.

Offit, who shares a patent on one of the vaccines, acknowledged to me that he would make money if his vote eventually leads to a marketable product. But he dismissed my suggestion that a scientist’s direct financial stake in CDC approval might bias his judgment. It provides no conflict for me, he insists. I have simply been informed by the process, not corrupted by it. When I sat around that table, my sole intent was trying to make recommendations that best benefited the children in this country. It’s offensive to say that physicians and public-health people are in the pocket of industry and thus are making decisions that they know are unsafe for children. It’s just not the way it works.

Other vaccine scientists and regulators gave me similar assurances. Like Offit, they view themselves as enlightened guardians of children’s health, proud of their partnerships with pharmaceutical companies, immune to the seductions of personal profit, besieged by irrational activists whose anti-vaccine campaigns are endangering children’s health. They are often resentful of questioning. Science, says Offit, is best left to scientists.

Still, some government officials were alarmed by the apparent conflicts of interest. In his e-mail to CDC administrators in 1999, Paul Patriarca of the FDA blasted federal regulators for failing to adequately scrutinize the danger posed by the added baby vaccines. I’m not sure there will be an
easy way out of the potential perception that the FDA, CDC and immunization-policy bodies may have been asleep at the switch re: thimerosal until now, Patriarca wrote. The close ties between regulatory officials and the pharmaceutical industry, he added, will also raise questions about various advisory bodies regarding aggressive recommendations for use of thimerosal in child vaccines.

If federal regulators and government scientists failed to grasp the potential risks of thimerosal over the years, no one could claim ignorance after the secret meeting at Simpsonwood. But rather than conduct more studies to test the link to autism and other forms of brain damage, the CDC placed politics over science. The agency turned its database on childhood vaccines—which had been developed largely at taxpayer expense—over to a private agency, Americas Health Insurance Plans, ensuring that it could not be used for additional research. It also instructed the Institute of Medicine, an advisory organization that is part of the National Academy of Sciences, to produce a study debunking the link between thimerosal and brain disorders.

The CDC wants us to declare, well, that these things are pretty safe, Dr. Marie McCormick, who chaired the 10Ms Immunization Safety Review Committee, told her fellow researchers when they first met in January 2001. We are not ever going to come down that [autism] is a true side effect of thimerosal exposure. According to transcripts of the meeting, the committee’s chief staffer, Kathleen Stratton, predicted that the IOM would conclude that the evidence was inadequate to accept or reject a causal relation between thimerosal and autism. That, she added, was the result Walt wants—a reference to Dr. Walter Orenstein, director of the National Immunization Program for the CDC.

For those who had devoted their lives to promoting vaccination, the revelations about thimerosal threatened to undermine everything they had worked for. We’ve got a dragon by the tail here, said Dr. Michael Kaback, another committee member. The more negative that [our] presentation is, the less likely people are to use vaccination, immunization—and we know what the results of that will be. We are kind of caught in a trap. How we work our way out of the trap, I think is the charge.

Even in public, federal officials made it clear that their primary goal in studying thimerosal was to dispel doubts about vaccines. Four current studies are taking place to rule out the proposed link between autism and thimerosal. Dr. Gordon Douglas, then-director of strategic planning for vaccine research at the National Institutes of Health, assured a Princeton University gathering in May 2001. In order to undo the harmful effects of research claiming to link the [measles] vaccine to an elevated risk of autism, we need to conduct and publicize additional studies to assure parents of safety. Douglas formerly served as president of vaccinations for Merck, where he ignored warnings about thimerosal risks.

In May of last year, the Institute of Medicine issued its final report. Its conclusion: There is no proven link between autism and thimerosal in vaccines. Rather than reviewing the large body of literature describing the toxicity of thimerosal, the report relied on four disastrously flawed epidemiological studies examining European countries, where children received much smaller doses of thimerosal than American kids. It also cited a new version of the Verstraeten study, published in the journal Pediatrics, that had been reworked to reduce the link between thimerosal
and autism. The new study included children too young to have been diagnosed with autism and
overlooked others who showed signs of the disease. The IOM declared the case closed and—in a
startling position for a scientific body—recommended that no further research be conducted.
The report may have satisfied the CDC, but it convinced no one. Rep. David Weldon, a
Republican physician from Florida who serves on the House Government Reform Committee,
attacked the Institute of Medicine, saying it relied on a handful of studies that were fatally flawed
by poor design and failed to represent all the available scientific and medical research. CDC
officials are not interested in an honest search for the truth, Weldon told me, because an
association between vaccines and autism would force them to admit that their policies
irreparably damaged thousands of children. Who would want to make that conclusion about
themselves?

Under pressure from Congress and parents, the Institute of Medicine convened another panel to
address continuing concerns about the Vaccine Safety Datalink Data Sharing program. In
February, the new panel, composed of different scientists, criticized the way the VSD had been
used in the Verstraeten study, and urged the CDC to make its vaccine database available to the
public.

So far, though, only two scientists have managed to gain access. Dr. Mark Geier, president of the
Genetics Center of America, and his son, David, spent a year battling to obtain the medical
records from the CDC. Since August 2002, when members of Congress pressured the agency to
turn over the data, the Geiers have completed six studies that demonstrate a powerful correlation
between thimerosal and neurological damage in children.

One study, which compares the cumulative dose of mercury received by children born between
1981 and 1985 with those born between 1990 and 1996, found a very significant relationship
between autism and vaccines. Another study of educational performance found that kids who
received higher doses of thimerosal in vaccines were nearly three times as likely to be diagnosed
with autism and more than three times as likely to suffer from speech disorders and mental
retardation.

Another soon-to-be published study shows that autism rates are in decline following the recent
elimination of thimerosal from most vaccines. As the federal government worked to prevent
scientists from studying vaccines, others have stepped in to study the link to autism. In April,
reporter Dan Olmsted of UPI undertook one of the more interesting studies himself. Searching
for children who had not been exposed to mercury in vaccines—the kind of population that
scientists typically use as a control in experiments—Olmsted scoured the Amish of Lancaster
County, Pennsylvania, who refuse to immunize their infants. Given the national rate of autism,
Olmsted calculated that there should be 130 autistics among the Amish. He found only four. One
had been exposed to high levels of mercury from a power plant. The other three—including one
child adopted from outside the Amish community—had received their vaccines.

At the state level, many officials have also conducted in-depth reviews of thimerosal. While the
Institute of Medicine was busy whitewashing the risks, the Iowa legislature was carefully
combing through all of the available scientific and biological data. After three years of review, I
became convinced there was sufficient credible research to show a link between mercury and the
increased incidences in autism, says state Sen. Ken Veenstra, a Republican who oversaw the investigation.

The fact that Iowa’s 700 percent increase in autism began in the 1990s, right after more and more vaccines were added to the children’s vaccine schedules, is solid evidence alone. Last year, Iowa became the first state to ban mercury in vaccines, followed by California. Similar bans are now (2006) under consideration in thirty-two other states.

But instead of following suit, the FDA continues to allow manufacturers to include thimerosal in scores of over-the-counter medications as well as steroids and injected collagen. Even more alarming, the government continues to ship vaccines preserved with thimerosal to developing countries-some of which are now experiencing a sudden explosion in autism rates. In China, where the disease was virtually unknown prior to the introduction of thimerosal by US drug manufacturers in 1999, news reports indicate that there are now more than 1.8 million autistics. Although reliable numbers are hard to come by, autistic disorders also appear to be soaring in India, Argentina, Nicaragua and other developing countries that are now using thimerosal-laced vaccines. The World Health Organization continues to insist thimerosal is safe, but it promises to keep the possibility that it is linked to neurological disorders under review.

I devoted time to study this issue because I believe that this is a moral crisis that must be addressed. If, as the evidence suggests, our public-health authorities knowingly allowed the pharmaceutical industry to poison an entire generation of American children, their actions arguably constitute one of the biggest scandals in the annals of American medicine. The CDC is guilty of incompetence and gross negligence, says Mark Blaxill, vice president of Safe Minds, a nonprofit organization concerned about the role of mercury in medicines. The damage caused by vaccine exposure is massive. Its bigger than asbestos, bigger than tobacco, bigger than anything you’ve ever seen.

It’s hard to calculate the damage to our country—and to the international efforts to eradicate epidemic diseases—if Third World nations come to believe that America’s most heralded foreign-aid initiative is poisoning their children. It’s not difficult to predict how this scenario will be interpreted by America’s enemies abroad.

The scientists and researchers—many of them sincere, even idealistic—who are participating in efforts to hide the science on thimerosal claim that they are trying to advance the lofty goal of protecting children in developing nations from disease pandemics. They are badly misguided. Their failure to come clean on thimerosal will come back horribly to haunt our country and the world’s poorest populations.

Even if the perfect vaccine did exist, without any side effects, it would still be a far cry from a magic bullet. People tend to overlook the fact that flu vaccines are manufactured before those viruses (virus stems) they are supposed to work against even exist.

Even mainstream studies have shown that during flu peak season, only 10% of infections that form in the upper airway can be traced back to influenza viruses. The statistic sounds reassuring and would make for great news if it weren’t for the epidemic hunters from the CDC, RKI or
WHO, who speak every year about another 10,000 flu deaths and urgently warn that only vaccinated people are protected from influenza.

Upon close examination of the data upon which their warnings are based, the question crops up: Are US flu death figures more PR than science? This is precisely the title of a study published in late 2005 in the British Medical Journal. Author Peter Doshi, of Harvard University (in 2006, Doshi switched to the Massachusetts Institute of Technology, MIT), provides a resoundingly decisive answer: US data on influenza deaths are a mess.

Doshi’s main criticism is that the CDC works under the assumption that 36,000 Americans die from viral flu each year—but they still owe us proof that an influenza virus really kills these people. Doshi’s conclusion: The CDC’s communication strategy is equivalent to marketing of fear. Several astute observers of the flu and vaccines critiqued the government’s promotional campaign urging the public to vaccinate against the flu by challenging the 36,000 annual death count the CDC attributes to the flu. Especially worth mentioning is the meta-analysis of the published flu vaccine reports by Tom Jefferson of the Cochrane Center, replicated in the British Medical Journal as well as a column in Red Flags by Edward Yazbak, a pediatrician. The findings of these 2006 articles are sobering: a major gap exists between evidence and public health policy.

The summary points of the BMJ’s meta-analysis are clearly alarming:
1. Because non-randomized studies predominate, systematic reviews of large data sets from several decades (meta-analyses) provide the best information on vaccine performance
2. Evidence from systematic reviews shows that inactivated vaccines have little or no effect on the effects measured
3. Most studies are of poor methodological quality and the impact of confounders is high
4. Little comparative evidence exists on the safety of these vaccines

The lead author Tom Jefferson concludes: The optimistic and confident tone of some predictions of viral circulation and of the impact of inactivated vaccines, which are at odds with the evidence, is striking. The reasons are probably complex and may involve a messy blend of truth conflicts and conflicts of interest making it difficult to separate factual disputes from value disputes or a manifestation of optimism bias, that is to say an unwarranted belief in the efficacy of interventions. In fact, the bottom line is that the CDC has not provided data to back up its claim about the number of deaths it attributes to the flu. The CDC appears to be acting on behalf of flu vaccine manufacturers, even as the evidence shows the vaccine to be worthless at best—or to be fatal at worst. A Vaccine Adverse Events Reporting System (VAERS) search performed on 10 October 2005 yielded three reports in the past two years of children younger than 23 months of age who died shortly after receiving a dose of influenza vaccine. No other vaccines were administered at the same time and all three children had underlying diseases.

We can only conclude that we are in the era of post-evidence-based medicine, states Vera Sharav from the Alliance for Human Research Protection in New York. Our public health policies are not even remotely evidence-based. Rather, our public health policies are faith-based decrees by government authorities—no better than voodoo medicine. Underlying this collapse of Western
medicine is the collusion between science and business. Our public health policies are currently shaped by corporate interests.

The CDC’s German counterpart, the Robert Koch Institute plays similar games with the statistics. They allege that in the winter of 2004 - 2005, 15,000 to 20,000 people died from viral flu in the country. But there is no proof to back up these statements. Rather, examining the data of Germany’s national office of statistics just nine people died of influenza viruses in 2004 (2003: 25; 2002: 10; 2001: 9). The picture painted by hospital statistics is just as undramatic: 12 deaths is a mere speck in comparison to the RKIs claim of 20,000 mortalities. Ask RKI to explain this extreme discrepancy and the institute answers that official statistic on influenza deaths underestimates the true influence of flu viruses, because very many influenza deaths are hidden in other diseases. For this reason, according to RKI, even the Statistisches Bundesamts data hardly reflects the true number of influenza deaths. But whereas the study showing concrete evidence that a virus was really at play, or was the single or primary cause in the cases where the RKI suspects a hidden flu virus? The RKI had no answer to this, even after repeated inquiries (see: Can We Trust Blindly The Figures of CDC, RKI, etc.?, Rapid Responses to Peter Doshis article in the British Medical Journal Are US flu death figures more PR than science?, British Medical Journal (Website), December 2005/January 2006).

Neither did we receive concrete studies from Berlins virus hunters to prove that 1) the flu virus declared a killer has been completely detected (purification and electron micrographs); 2) the virus, insofar as it does exist, has lethal properties; and 3) all other factors (nutrition, toxins, etc.) can be ruled out as primary or major causes of the so-called flu victims death.

The RKI says it arrived at the 15,000 to 20,000 flu deaths by applying an internationally recognized and peer reviewed calculative method. But whether a calculation makes sense cannot be determined by the fact that it is recognized and has been verified by other researchers, but only by being verified by independent technical experts. We wanted to do this, but so far it has not been possible. In December 2005, the RKI did agree to send us their detailed calculations by the end of January 2006 at the latest; we have yet to receive them. Yet the RKI should actually have the calculation at hand.

The RKI also claims it is often the case, that influenza death figures are estimated values. And in this regard as well, they agreed to send us the documents that support this by the end of January 2006. But unfortunately, we have not yet received a single document from the RKI. One thing is certain: contrary to what the RKI told us, in its database of significant papers and statistics, the RKI does not explicitly say that only estimated values are available. This is true on their website, for instance, where influenza mortality figures are listed, and in a press release from late 2004.

The RKI identifies the influenza work-group as the source of their influenza data. The AGI was founded by the pharmaceutical industry in 1991, and receives financial support from four vaccine manufacturers. So, if the RKI relies on an organization funded by the pharmaceutical industry, how can the institute make sure that published data is absolutely sound?

It would be wise to ask the same question of the German vaccine committee STIKO a part of the RKI system. STIKO Chair, medical professor Heinz-J. Schmitt, is also on the Board of Directors.
of Foundation for Preventive Pediatrics), a children’s health foundation which in turn works closely with and is funded by pharmaceutical companies like GlaxoSmithKline and Chiron-Behring. Schmitt additionally functions as consultant to the GlaxoSmithKline project Gesundes Kind (Healthy Child), which plugs protective vaccinations.

To be able to evaluate whether RKI can still act independently of the pharmaceutical industry, we requested that the institute disclose all the ways their scientists are remunerated (lecture fees, research grants, etc.). By their scientists, we mean the ones working for the RKI or for other institutions directly subordinate or integrated into the RKI.

But to date, we have not received a response to any of these questions. In any case, it is certain that several STIKO members cultivate close relationships with Big Pharma or are active for pharmaceutical companies, including the major ones like GlaxoSmithKline. It is also telling that the RKI, as Focus magazine reported in a rare critical article on epidemic authorities, were confronted with the revelation of a corruption case in early 2006, which cast a very negative light on the highly esteemed institution.

Social researcher Friedrich T. [full surname not mentioned], who had worked as a top official at the RKI, was sentenced by the district court Berlin-Tiergarten to six months in prison and a fine of € 3,000. In late 1998, T. had internally proposed awarding the contract for a reputedly extremely important AIDS study (RKI Sentinel) to a private polling institute by the name of Images. And indeed Images bid for the study worth 396,000 German marks (approximately $200,000) was accepted. Two months later, an Images employee turned over 10,000 marks in cash to T. The presiding judge saw the elements of corruption here, as she explicitly declared this a not unserious case. During the trial, the judge had declared that there were evidently a few alarming interconnections at the RKI. She was convinced that more was known at the institute than came out in the trial. The final verdict also stated that the court cannot resist the impression that here on a large scale, the RKI has been used as a good source of money.

The company Images functioned namely only as a dummy firm for the identically staffed and located Intersofia GmbH (Ltd.), whose founder and sole shareholder is none other than RKI official T. Two Intersofia employees had founded Images expressly for the purpose of landing the AIDS study contract, since T. couldn’t directly hand the contract to his own company Intersofia. T. penned not only the service description for the RKI Sentinel but also Images offer.

On 3 November 1998, T. proposed the dummy company as contractual partner, but Images was not founded until 15 November, and five days later, ministry director Reinhard Kurth personally signed the contract.

Focus magazine is completely correct in writing that T.s corruption case had turned into a worst-case scenario for Reinhard Kurth as well. Kurth had evidently also lied to the public. The RKIs press office and even the RKI president declared to know nothing of any possible conflicts of interests for T. at the time the contract was awarded. But this claim is impossible. In her verdict, the judge cited the testimony of a certain Wolfgang Kurtz, who was Director of Central Administration at the RKI during the time in question (first half of November). According to Kurtz, the epidemic authority’s Research Council, which was responsible for awarding the
contract, were fully aware that T. was doing the AIDS study with his old mates. Additionally, the researcher’s financial sleights of hand had been a constant gossip topic at the institute for years. By the end of 2000, top management had detailed information on the Intersofia/Images scam. An employee of T.’s private company had filed a disciplinary complaint against her boss with the RKI, revealing details about the scheme. A whole year later, Kurth declared that internal clarification of the accusations was proving to be difficult and time-consuming. But in T.’s trial, the district attorneys simplified this allegedly complex issue. The accused had seen the RKI simply as a sort of self-service shop. Perhaps he thought he was invulnerable.

Not only did T. have good contacts at the top of the Federal Health Ministry, he also collaborated very closely with his superior, no less than Barbel-Maria Kurth, RKI department head, and the president’s wife.

T. also took care of a particularly awkward assignment for his boss. Mrs. Kurth had tried to safeguard GDR scientist Michael Radoschewski’s career for many years, after it had gone into a tailspin post-reunification. Because of his former Stasi (East German secret police) activity, he could not get a steady job in unified Germany’s health administration. Mrs. Kurth, herself a former GDR student, helped with labor contracts, and ultimately accommodated him in the firm Images, T.’s dummy company. Radoschewski even worked on the AIDS study. In this way, the RKI continued paying his salary indirectly.

The AIDS study, financed to the tune of approximately $200,000 worth of tax dollars, was incidentally not published. T. and his Images troupe had sunk the project. Images former Managing Director, Liane S. appeared as a witness in the trial. The judge dismissed her attempts at exoneration, calling them lies. But why would Mrs. S. have said anything bad about T. and his insider dealings? S. now works at the RKI-in Mrs. Kurths department.

As has repeatedly been portrayed in this book, there is certainly no reason to assume that such conflicts of interest and corrupt activities are the exception, and to suppose that, on the whole, everything is just fine. Transparency Internationals Corruption Annual Report 2006 is worth another mention. The report was presented to the public in May of the same year, and unequivocally says that waste, fraud and corruption have eaten into the local public health service and annual damages are at least € 24 billion.

This rarely publicly addressed mismanagement can only be fixed with great difficulty because the industry in question is run by powerful corporations and its allies—including decrepit government organizations that lack transparency and federal oversight. Transparency International clearly awards chief responsibility for this mess to the pharmaceutical industry, which forges studies, influences authorities, suppresses risks and undermines alternative health and self-help groups. 40% of medical studies from 2005 were demonstrably faked or manipulated by sponsors.

Politics has yielded to health lobbyists for too long, says the watchdog organization. Health service bodies governed by public law at the Federal State level have been left to their own devices for too long. It is time to look for a means of compulsory accountability for everything.
This includes, above all, the best possible transparency for contributors and taxpayers. Often though, nothing happens, because doctors, researchers or pharmaceutical lobbyists have strong connections to politics. Corruption fighters also demand a radical professionalization among the health care system players, especially the insurance companies, the panel doctors associations and government institutions in order to make their decision-making processes more transparent. There must also be a stronger enforcement of the law, in order to ban bad doctors from the profession.

Transparency International also recommended requiring disclosure of financing and relationships to sponsors, as well as the registration of all clinical trials. To avoid deadly mistakes, the health care field should not be allowed to purchase medical experts for their pharmaceutical studies and consequent marketing. Additionally, there needs to be legal regulations for health insurance companies to maintain accountability and public safety. The establishment of specialized district attorneys would also be sensible.

But structural corruption cannot be tackled simply with new laws, reforms and better law enforcement, according to the anti-corruption organization. A culture has to be generated that outlaws fraud in medicine. It is immoral and indecent to make money from a system that is putting an increasing strain on people with low incomes, and allow increasing gaps in a comprehensive complete medical care, through faulty calculations.

It would be extremely helpful if the media—the States (self-declared) fourth power—would turn itself again to its true task and consistently try to bring the structural corruption in the health service to light, instead of playing henchman to Big Pharma.

Today, jubilation is expressed by both orthodox science and the mass media about the recently developed vaccine against the human papillomavirus (HPV) assumed to cause cervical cancer. The HPV vaccine is being marketed heavily, especially for use in girls 9 - 15 years of age. In the literature, we read that the vaccination has been proven to be the most efficient and logistically feasible preventive intervention against cervical cancer. And the vaccine makers promise an almost 100% protection, according to a lead story in the Frankfurter Allgemeine Zeitung written by the head science editor himself, headlined: Vaccinating Against Cancer-In the Drugstore a Dream Comes True.

According to one of Germanys most important daily newspapers, we now see the start of a new epoch. Heading the march into a new golden age is pharmaceutical company Sanofi Pasteur MSD, with a new vaccine called Gardasil. The announcements by the manufacturer could be dismissed as typical pharmaceutical industry pursuit of giant markets, profits, power and prestige. Yet, en masse, physicians and scientists have joined the chorus, which speaks to a paradigm shift. All are gushing about the potential to abruptly stanch one of the worst villains for women with only three harmless injections. The results of the [vaccines] approval studies are so convincing that by now there is no limit to euphoria.

Again, the news sounds more than good. But, before we uncork the champagne, should we really believe the promises of this pharmaceutical giant, brush aside all the conflicts of interests today’s
biomedical science and forget all the previous empty promises made by even the most prestigious researchers?

In order to clarify this, we approached one of the relevant institutions from which all these predictions, assertions, and claims stem from: The German Cancer Research Centre DKFZ. What we asked for was:

1. A solid study proving the existence of a human papillomavirus, in short HPV (including a description of the purification and isolation of the particle as well as the characterization of the full genome and the mantle, plus an image done by electron microscopy)
2. A solid study proving beyond doubt that HPV causes cervical cancer
3. A solid study showing that non-viral factors such as nutrition or chemical toxins alone or in combination can be excluded as possible (primary) causes for cervical cancer
4. A solid study demonstrating conclusively that the vaccinations entering the market are safe and effective.

Indeed, as response we received a wonderful literature list, as the DKFZ declared, on which are several studies being mentioned addressing at least items 1, 2, and 4. Unfortunately, missing from the list was a study proving item 3, that non-viral factors such as nutrition, pesticides, stress, etc. alone or in combination can be excluded as possible (primary) causes for cervical cancer. Interestingly, even the medical establishment itself identified non-viral factors such as smoking or the use of oral contraceptives which are viewed as relevant co-factors in the development of cervical cancer. And there is no proof that these factors could not act as primary factors.

In this context it is also worth mentioning that in the search for the causes of cervical cancer the fact is being disregarded that up to 80% of all woman at least temporarily shall contract this so-called papillomavirus during her life, but in 80% of these women the virus just disappears after a while. That is to say that only in 20% of the cases the doctors register (with their test methods) a continuing infection that according to orthodox researchers shall carry the risk of causing cervical cancer.

And according to Lutz Gissmann from the DKFZ in Heidelberg as a matter of fact much Jess than 1% of these infected women come down with cancer. We just don’t know why most women are able to cope with the virus, Gissmann concedes. That means-assuming that we can believe the methods of virus detection—in most cases of cervical cancer there is a positive HPV test, but in only a tiny minority of cases is cervical cancer found.

There must be other factors responsible for the development of cervical cancer. And there is obviously no proof that these non-viral factors cannot play the major or primary role. And so it is not really surprising to hear from one of the leading established cervical cancer researcher, Matthias Durst from the University of Jena, that the infection with the papillomavirus alone still does not cause cancer. The tumor is said to grow not until there are genetic changes on the chromosomes causing this accretion. But here we have the same problem: there is not a single study proving that a (papilloma)virus initiates these genetic changes or chromosomal alterations. But let’s step backwards again and ask: can we really believe the methods of virus detection? As mentioned before, the DKFZ sent us this wonderful literature in which there are two studies
both conducted by zur Hausen et al. that they claim serve as proofs for the first isolation of specific HPV from cervical cancer tissue. But a closer look into these trials reveals that actually there is no such kind of proof, says Canadian biologist David Crowe. For example, the first of these two papers published in 1983 in the journal Proceedings of the National Academy of Sciences: A Papillomavirus DNA from a Cervical Carcinoma and Its Prevalence in Cancer Biopsy Samples from Different Geographic Regions, Jacks the following critical issues: 1. It is not clear where the cloned DNA of the presumed virus comes from. But without knowing the origin of the DNA it is impossible to prove that a virus is there.

2. A large number of tumors were screened without success, increasing the possibility that this discovery of one tumor with this DNA is just a coincidence. The cancer establishment is always talking about the high correlation between HPV screening of people suffering from cervical cancer. But it should be noted that particles called HPV are quite common, so to say that HPV is usually found in people with cervical cancer might not mean much.

3. The authors use the term non-stringent conditions which probably means that hybridization (formation of base pairs between complementary regions of two strands of DNA that were not originally paired) occurred with less than a perfect match. That is to say, the two DNAs they were using were not identical. Of course, they will just say that viruses mutate rapidly, Crowe points out. But this is pure speculation.

4. They extracted DNA and hybridized that with known HPV samples-but they got less than a 0.1% match. Because of this they declared that it was a new species, as opposed to declaring that they had pulled out DNA that had nothing to do with HPV at all.

5. So now with this new DNA, with little relation with other HPV DNA, they declare that because it matches 11 out of 18 cervical cancers, it proves that the cervical cancers contain this new HPV. Yet they haven’t proved that this is a virus at all!

We approached the DKFZ twice with our points of criticism asking for clarification. But we didn’t get any response. That raises the important question: Why should a woman undergo a PAP smear or an HPV test supposed to detect papillomavirus-DNA (not even for the detection of the virus itself! ) if (1) there is no scientific proof of this virus and (2) even the cancer establishment admits that the papillomavirus does not cause cancer on its own?

Apart from this, critics of the cancer orthodoxy emphasize that the PAP smear test developed in 1928 by the Greek medical doctor George Papanicolaou is practically meaningless. The test just rests on the evaluation of cell changes found in smears taken from the uterine orifices that are said to cause cancer. But this is pure theory, and the test just classifies too many women as being at risk of getting cervical cancer.

Established cancer scientists such as Diirst don’t agree and counter that a negative PAP smear test result would suggest unerringly in 99.6% of the cases that a woman did not come down with a precancerosis (tissue alteration that is associated with a higher risk of becoming a malignant degeneration) or cervical cancer.
Sounds very good, but this magnificent promise is qualified if we take a look at the statistics. In Germany, for example, every year around 7,000 women fall ill from cervical cancer, that is to say 0.017% of the 40 million women living in Germany. This means, 99.983% of these women do not develop cervical cancer. In other words, cervical cancer is a very rare disease, and it is very easy to achieve 99.6%-safety, not from the PAP smear test, but from the statistic alone.

Furthermore, the PAP smear test has a high error rate. It happens, for example, very often that sick cells are overlooked because simple inflammations canvas the sight at mutated cells. In one examination at the University of Hanover, the screening tests yielded 86 suspected cases, but posterior control tests could confirm only 46 of the suspected cancer diagnoses. This is an error rate of almost 50%. Karl Ulrich Petry, gynecologist and one of the leading researchers of the study: Cervical cancer screening sometimes is like trying to nail jello onto the wall. The collected data is not really reliable.

Nevertheless, in the USA alone, every year around 200,000 women have their uterus removed, many of them to prevent cervical cancer. But in fact only 14,000 American women come down with cervical cancer each year. That is to say, tens of thousands of women in the United States are being operated—or shall we say: garbled—unnecessarily or at least hastily. The reason is that the PAP smear test is not searching for early forms of cervical cancer cells, but for pre-forms which very often degenerate by themselves or stay innocuous.

In 2003 the British Medical Journal published a study about the outcomes of screening to prevent cervical cancer. And the results are not encouraging: around 1,000 women need to be screened for 35 years to prevent one death; 150 of these women will receive a stress-causing test result, and 50 women will go through cancer treatment with all its highly toxic side effects. For each death prevented many women have to be screened and many are treated who would not have developed a problem, writes Angela Raffle, the leading author of the trial. In other words: There is just no scientific proof for the effectiveness of the screening tests and their collateral side effects (stress, operation, medication) are more than worrying.

The same holds for the HPV tests, introduced in Europe some years ago. They are considered and promoted to leading to much more reliable and exact cancer checkups. But the lack of an HP-virus proof alone makes these tests worthless. In addition to this these tests entail the big risk of classifying even more women, who will most likely never get a tumor in their uterus during life, as endangered of getting cervical cancer-leading to even more needless operations and medications. In this context let’s not forget the fact that only around 0.1% of the women said to be infected with HPV fall ill with cervical cancer—so in consideration of this extremely low frequency it remains an enigma how established cancer authorities can speak at all of a high of a connexion between cancer and an HPV.

Nobel laureate for Medicine Sir Frank Macfarlane Burnet warned us against jumping to any conclusions about a potential link between cancer and viruses in 1971, in the book Genes, Dreams and Realities: In the last dozen years there has been a great concentration of research on the viruses which can produce cancer or leukaemia of mice, hamsters, and chickens. There is no doubt at all about the genuinely malignant character of the tumors which are produced but so far
there is no convincing evidence that any human tumor is virus-induced. One must be definite that despite ten years intensive study the virus theory has established itself as nothing further than speculation. There may be almost a majority of younger cancer research men who think it likely that eventually cancer will be shown to be due to the action of slow viruses which in the great majority of people persist without any visible effect. To me this is an unjustifiable and unscientific act of faith based on a failure to understand the significance of the work on viruses of laboratory animals.

My great objection to the hypothesis that any human cancer is a direct result of virus infection is my inability to conceive of a selective process in nature that could be equivalent to the laboratory procedure. Considering the extreme rarity of cancer in wild animals I can see no way by which an ability to induce cancer could favor the survival of a virus species. Neither can I see anything in human biology which could have power to evolve human cancer viruses; except by deliberate human effort directed to such an end. I believe we can forget about the possibility of any of the common forms of cancer being of virus origin.

Chapter 8
If we visualize the facts about HPV-no proof for virus detection; no proof for HPVs pathogenicity or for HPV being the primary, let alone single cause of cervical cancer; non-HPV causation omitted; only 0.1% of the so-called HPV-infected women coming down with cervical cancer-one must conclude that the vaccinations entering the market cannot be safe and effective.

All the worse that the US drug approval agency FDA appears to have learned nothing from recent catastrophic disasters due to the agency’s approval of unsafe drugs-such as Mercks anti-inflammatory drug, Vioxx. The FDA hastily approved Mercks HPV vaccine Gardasil which is designed to prevent cervical cancer and genital warts in sexually active women. However, the vaccine has not been proven safe and effective in clinical trials, either. The trials are being criticized for using a placebo containing aluminum adjuvant (whose adverse reaction profile makes the vaccine appear safer than it is), rather than using a non-reactive saline solution placebo.

Here’s how: the vaccine triggered adverse event reports in 90% of the test subjects within 15 days-hardly an indication of safety. However, the controversial placebo formula triggered 85% adverse event reports. How does the FDA know what long-term adverse effects the vaccine might produce? The more so as Gardasil comes along with heavy side effects ranging from reddening and swellings around the injection spot, fever, hives, arthritis, and even death.

It seems as if the medical establishment learned nothing from the disastrous DES (diethylstilbestrol) effects on the daughters of women who took the hormone during pregnancy triggering cancer and genital deformities. This is a particular concern because the HPV vaccine is being promoted for use in girls between 9 and 15 years of age. But the vaccine has never been tested for girls in this age group who are in a most sensitive phase of their development. Vaccinating these girls and young women has to be called negligent. Not least because not even the minimum protecting antibody concentration is known, nor the duration of the protection of the vaccination nor the necessity of booster inoculations.
Sure, the DKFZ and other established cancer institutions never tire of saying that the vaccines protective effect is 4 to 5 years, but this is nothing more than pure and unfounded speculation that benefits the marketing of a medical substance that is promising very high profits for the pharmaceutical giants making it.

National Vaccine Information Center president, Barbara Loe Fisher, says Mercks pre and post-licensure marketing strategy has positioned mass use of this vaccine by pre-teens as a morality play in order to avoid talking about the flawed science they used to get it licensed. This is not just about teenagers having sex, it is also about whether Gardasil has been proven safe and effective for little girls.

Lets not forget that the idea of immune therapy for cancer is 100 years old. Paul Ehrlich already postulated that one can use immunity to fight against cancer. In the April 2005 issue of Nature Medicine a trial vaccine is described that for the first time ever is supposed to be able to extend the life expectancy of patients with prostate cancer. But Ehrlichs trial and all other attempts to make a virus-disease out of whatever type of cancer was, are and always will be hopeless ventures.

The reason is as simple as it is evident: The cancer cell does not contain new genetic material-but the immune system still only recognizes foreign material, as cancer researcher Peter Duesberg points out. If mutated genes could activate the immune system, then we all would be long dead, because the immune system would kill cells daily en masse. In actuality, ordinary gene mutations are channeled through the body under the radar screen of the immune system. The topic is often revived, but always it turns out to be a false alarm.

If HPV were the cause of cervical cancer, then it must be transferred also from the female partner to the male partner. But even if we assume that the HPV tests indeed measure HPV, it is still fact that HPV is practically not detectable in men, nor does it induce health problems in males. This speaks strongly against an infectious cause of cervical cancer, says gynecologist Christian Fiala. Furthermore, a PAP smear test being conducted badly in many cases results in a resection of uterine orifice tissue exactly where the tissue degenerations are. After the tissue is cut out, further degenerations are rarely observed. But if all this is caused by an infection, it couldn’t be treated surgically.

When the science becomes politicized-whether from the conservative right or from the liberal left-we cannot trust anything that’s being said. Absent scientific evidence demonstrating the safety of the HPV vaccine, there is no guarantee that this will not prove to be a disaster for the next generation. We can only conclude that we are in the era of post-evidence-based medicine, states Vera Sharav from the Alliance for Human Research Protection in New York. Our public health policies are not even remotely evidence-based. Rather, our public health policies are faith-based decrees by government authorities-no better than voodoo medicine.

Epilogue
Even if the medical establishment particularly or exclusively recommends vaccines and antiviral medicines in the fight against disease like the flu/ the determinants of health lie in large part...
outside the medical system, writes Thomas McKeown, professor of social medicine, in his work "The Meaning of Medicine. The only effective way to combat influenza or other diseases (baselessly connected to viruses), while also safeguarding our hearts, lungs, livers and brains, is to strengthen our immune systems.

This unquestionably includes avoiding contact with chemical toxins. But in our virus mania, more than 100,000 industrial chemicals are disregarded as culprits. They exist, everywhere, whether in children’s toys, computers, textiles, cosmetics, electronic appliances or foods. And most of these substances have never been rigorously tested to investigate how much damage they can do to human health and nature as a whole, in the short and long term.

Children already have a dangerous cocktail of chemicals in their blood: a mixture of potentially highly dangerous substances, which little by little can accumulate dangerously in the body. Where are the health authorities that, for example, stand up for a War on Toxins, willing to liquidate hundreds of billions in assets, and following the precautionary principle-prohibit chemicals when their harmlessness has not been scientifically proven?

The same question crops up with genetically modified foods, without which the world has done just fine for billions of years. Why, then, should this be any different today? Ultimately, they only serve to secure profits for agricultural and foodstuff groups. But scientific investigations show they hold potential dangers that nobody can really estimate. In late 2005, the Australian Commonwealth Scientific and Research Organization (CSIRO) broke off their experiments with genetically modified peas after test mice had serious reactions (particularly with lung diseases). It could absolutely be assumed that something in the peas had compromised the immune system, says Thomas Higgins, assistant director of the CSIROJ.

Earlier, experiments with rats fed MON863, a genetically altered corn, had shown that MON863 led to alterations in blood count and the animals organs. By early 2006, the EU had still not succeeded in achieving a majority against the controversial foodstuffs approval. But, MON863 has already been authorized as animal feed throughout the EU.

Unfortunately, avoiding such toxic substances won’t be easy. This is all the more reason to do as much as you can to keep your health up to scratch for as long as possible. In this respect, much too little attention is still paid to the intestine. We have already addressed this, but here we would like to do it again, for its significance to the human body is still often underestimated, writes Wolfgang Kruis, medical professor and intestinal expert in Cologne. With its 200 m2 large, microbe-saturated intestinal flora, the intestine presents by far the largest immune system in our bodies.

Just how fit this intestinal flora is, is in turn influenced by a whole range of factors—for instance, to what degree and over what period we expose our bodies to stress, lack of exercise, toxic drugs like cigarettes and alcohol, and above all poor nutrition.

In general, nutrition is attributed a central role. Consumption of too much meat, fish, cheese, white bread and refined sugars can cause vitamin deficiencies and produce numerous diseases, including many flu-like symptoms such as headaches or sinus infections, lack of drive, bone
atrophy and depression. Often, too few enzymes the ignition sparks of life-are ingested, something that can compromise numerous body functions and also weaken the immune system. Every human organ, tissue and cell functions with the assistance of enzymes. Eating, sleeping, thinking and even feeling are accompanied by enzyme activity.

There are said to be 40,000 of these protein molecules. We produce some of them ourselves, but many must be consumed through food. And many environmental toxins act as enzyme inhibitors, like carbon dioxide or heavy metals like mercury and cadmium. Above all, enzymes are extremely heat-sensitive. At 45 degrees, they lose their effects. This means that in cooked foods and also in pasteurized and processed foods, there are no more effective enzymes. They should best be consumed in the form of fresh fruit and vegetables.

Selenium or zinc deficiencies can often exist, which are likewise associated with damage to the immune system. Plenty of selenium is found in coconuts (810 micrograms or 10g per 100 g or 3.53 oz.), for instance, while Brazil nuts contain a lot of zinc (4,000 micrograms or 1g per 100 g or 3.53 oz.). Eating whole foods, and even better, having a holistic view (instead of pill-popping), is sure to set the immune system on the right path. Let’s say we knew all the contents of a pear, writes Angelika Langosch in her dissertation: Influence of Nutrition, Particularly Raw Foods, on Intestinal Flora and Infection Defense. Then, the respective amounts of all these ingredients would merely produce a mixture of these substances in a watery solution, but not a pear. A food is more than the sum of its parts.

The idea that what nature has provided us with could be replaced by preparations like vitamin, mineral and enzyme tablets, artificial flavorings, designer food from the chemistry labs and a few laxatives, as well as artificial air from the air conditioner and a sedentary life spent in automobiles and in front of computer and televisions, ultimately only helps to secure the profits of various giant corporations. These things do not make us healthy. If this were true, then there wouldn’t be so many sick people-and affluent societies are primarily affected by chronic diseases like allergies, diabetes, heart disease, osteoporosis and cancer. In contrast, diseases like cancer are virtually unknown in wild animals, even in elephants, which have approximately the same life expectancy as humans, or in whales, which can live for more than 200 years.

The idea that artificial products could replace nature and maintain or even manufacture health is merely due to a Cartesian worldview (tracing back to Rene Descartes, 1596 - 1650), in which the modern individual’s thoughts are ensnared.

Ultimately, this viewpoint reduces living beings to machines that can be fueled artificially, with pills thrown in from time to time, and, if necessary, rigged with substitute replacement parts. And so we carry over principles that have been successfully applied to inanimate nature to living beings, writes McKeown. This model would long have been rejected if it seriously contradicted experience-if humanity, then, finally realized it had come to a false conclusion. We mistakenly believe that the retreat of infectious diseases-the main reason for improvements in public health-is substantially due to advances in medical science, as McKeown point out. In truth, the vast improvement to public health [only] profited a little from the contributions of science and technology. Instead, the advances can be traced to simple but momentous everyday discoveries:
for instance, increases in food production through conservation of soil fertility, or hygiene improvements.

Reports on certain primitive peoples also show that one can live very healthily without the blessings of the pharmaceutical industry. In his diary, the Frenchman Jean de Lery admiringly recounts the wild Americans with whom he lived in the mid-16th century, in what is now Brazil: They are a great deal healthier than us [Europeans] and suffer less from diseases. It is very rare to see lame, one-eyed, or deformed people among them. Not few of these people attain an age of one hundred to 120 years, and only a few have white or even grey hair. Lery is praised by specialists for the objective style of his descriptions. The famous ethnologist Claude Levi-Strauss even paid him the compliment of the modern scholar in his book Tristes Tropiques.

Besides Lery, all of the 16th century’s other travelers were downright amazed at the vivid beauty and stable health of the native men and women, who cultivated a totally simple lifestyle and ate natural foods (so unlike ours today which, thanks to over-industrialized chemical farming often taste like cardboard and are deprived of important nutrients). Lery gushed poetically about the pineapples grown in the wilderness, whose strong strawberry scent one could already smell from afar and which melt in your mouth and are naturally so sweet that they cannot be bettered by any of the jams we usually have in Europe.

And so the people of the Renaissance ultimately observed with amazement that their own antique ideal had found its realization overseas in these native men. In our overmedicated, high-tech and overworked society, the idea that health can be easily had without the medical and food industries with their medicines, vitamin pills and dietary supplements may sound strange for many of us nowadays. And one might wonder: if everything that politicians, researchers and journalists sell us as truth is actually false, how could all the mistakes go undiscovered for so long? Shouldn’t the conclusions outlined in this book have gone off like a bomb a long time ago?

The primary reason this has not happened is that it’s too simple for many people to imagine. Intelligent researchers have chosen to overlook it for decades. It is too shocking for us to believe that we’ve been lied to by the very people charged with safeguarding our health. Above all, none of them are interested in these simple pursuits. Doctors would have to go on a totally different path in order to achieve fame and honor (or abandon such a goal altogether and change their definition of success). Medical statisticians would be sawing off the very branch on which they perch. Pharmaceutical companies would have to completely overhaul their bottom line-obsessed industry and actually invest resources in developing effective medications instead of ones that do nothing, harm or even kill.

Ultimately, the only individuals who would profit from this would be patients. But first, they have to educate themselves and take back control of their own bodies. And with this book, we hope we can make a contribution to this pursuit—for a better, more peaceful and healthier future for our beloved planet and all its habitants.