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AIDS, Hepatitis C, BSE:
Infectious or Intoxication Diseases?

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If you are willing to believe the medical as well as the general press, the world today is again and again beset by new big epidemics. First AIDS, then hepatitis C, now BSE. These new plagues differ from the plagues of the past in one respect: The number of affected people is relatively small.

While the old plagues annihilated whole towns, the number of people who actually fall ill with the "new big plagues" is very low. In the case of AIDS there are about 2000 "new infections" (HIV antibody positive) every year, and 600 deaths [in Germany]. Hepatitis C hasn't led to a significant increase of liver cirrhosis, and regarding BSE, we still don't have even one clinical case in our country, while the press has been talking about BSE crisis or epidemics for weeks.

The epidemic-like character of these diseases is generated by a molecular biological phenomenon, namely so-called test explosions. Today molecular biology is capable of detecting the smallest quantities of DNA or RNA with the PCR (Polymerase Chain Reaction) and able to produce antibodies against it. The connection between what has been isolated in humans or animals, and the presence of clinical symptoms, is a mere hypothesis. This is perfectly illustrated in BSE, where a testing epidemic has also taken place now, and still not one clinical symptom (a mad cow) has appeared [in Germany].

Because the symptoms are often missing, they must proclaim endless latency periods, up to 55 years (between infection with the "BSE pathogen" and developing the new variant of Creutzfeld-Jakob-Disease). But let's start with AIDS, the first of the big new plagues.

AIDS

AIDS manifested itself in the early 80s in San Francisco and affected only homosexuals, who at the age of [about] 30 developed PCP (Pneumocystis Carinii Pneumonia) and in part died of it. These first patients, whose cases were published by Dr. Gottlieb, had two things in common; they were homosexual and they were heavy drug addicts (cocaine, amphetamines, Amyl nitrites).

Amyl nitrite is a sex drug, that is almost exclusively used in homosexual community and which is induced in large quantities via inhalation. Nitrates are, testable in animal research and in lymphocyte cultures, immuno-toxic and cytotoxic as well as cancerogen (Source: NIDA, National Institute of Drug abuse). Before the acronym "AIDS" was born, the same thing had the name GRID (Gay Related Immune-Deficiency). During the first years science assumed a lifestyle disease, because it was obvious that AIDS only occurred in certain communities (homosexuals who lived the "fast-lane-lifestyle"). In 1983 the US health minister proclaimed on a press conference that a US researcher had discovered a retrovirus which was the probable cause of AIDS. The next day all papers wrote that a US researcher had discovered the cause of AIDS. They had forgotten the word "probable"... since then all research and therapy has taken place only from the view of the virus hypothesis. In other words, for the past 17 years the question has been researched: how HIV does cause AIDS; the question IF HIV does cause AIDS must not be asked anymore.

Years later, Kary Mullis, Nobel laureate in chemistry 1993 and inventor of the polymerase chain reaction, needed a reference for "the generally known fact" that HIV was the cause of AIDS. While working on a project, he became aware that he didn't know a scientific reference for the statement he had just written down: HIV is the probable cause of AIDS. So he asked the next virologist at the table after that basic paper. The virologist told Mullis, he wouldn't need a reference in this case; after all, everyone knows that HIV leads to AIDS. Kary Mullis disagreed and thought such an important discovery should be published in some paper. He learnt soon that it was impossible to find such a paper. Instead, he was pointed to the press conference of 1983 over and over again.

One day, he got the opportunity to talk to Luc Montagnier from the Pasteur Institute, the [claimed] discoverer of the virus, during an event in San Diego. HE should know the answer. Confronted with Mullis´question, Montagnier said: "Why don't you cite the report of the CDC (Centers of Disease Control)?" Mullis answered: "This report doesn't address the question whether or not HIV is the cause of AIDS" - "Right", Montagnier admitted, "but maybe you could cite the SIV study (Simian Immuno-deficiency Virus, which is very similar to HIV)." That paper didn't convince Mullis either, because the monkeys developed different diseases, also because the virus wasn't the same one, and thirdly, because the paper had been published only a few months before. He looked for the original paper that should demonstrate in whatever form that HIV was the cause of AIDS. At that point, Montagnier's answer consisted of running away, to greet a group on the other side of the room.

I had a similar experience this year [2000] in South Africa on the AIDS Advisory Panel, which had

been initiated by President Thabo Mbeki. Mbeki had invited 33 scientists from all over the world in order to shed light on the AIDS problem in his country. Among these panellists were 22 scientists who believed in the virus hypothesis. Furthermore, there were 11 so-called dissidents (which I belong to), who cast doubt on the virus hypothesis and rather assume that AIDS in Africa is the result of increasing poverty. In the developed countries AIDS is the result of drugs, and above all the result of the therapy against AIDS (AZT).

I asked Montagnier what convinced him that AIDS is caused by a virus. Montagnier answered that over the years apparently an effective treatment has been developed, and this was proof enough for HIV leading to AIDS. In other words, the virologists have no virological arguments for the theory that HIV leads to AIDS. Instead, they get the proof for their hypotheses from physicians, who give a positive feedback by saying "Of course AIDS is a viral disease that responds to antiviral treatment." However, we doctors treat HIV-positive patients basically differently from if they were HIV-negative. From shingles to apoplexy, HIV-pos. Patients are given a lifelong antiviral treatment, or we treat them usually without any clinical illness, only on the basis of surrogate markers like CD4-cells and viral load, which (latter) can be measured via PCR (the method invented by Kary Mullis). Mullis on his method: "It is nonsense to amplify something that is detectable only by PCR and which is practically zero; it will still be close to zero."

Now in Africa, on the panel, it also became obvious that the initial dose of 1500-mg AZT (1987) was much too high. In other words, it became clear that the situation of the patients then wasn't improved by this high dosed therapy -but worsened. The high mortality of the AIDS patients at that time was not too striking though, because it was the general expectation that AIDS patients will die fast and young.

The problem of the therapy was and is that it is extremely immuno-suppressive itself. AZT was developed in the 60s specifically as a chemotherapy against cancer. But wasn't used then due to severe toxicity. However, a few pre-studies had been carried out, so that the substance could be used in the 80s. Then AZT was tested in a placebo-controlled study in 1987. This study was cancelled after four months, because at that time it looked like the patients in the treatment group would derive benefit from the therapy.

The publication in NEJM led to the world-wide use of 1500-mg AZT for AIDS patients and HIV-positive people. The reason for the early cancelling of the study was the unbelievable pressure from lobbying groups who hoped a cure was found. But afterwards mortality in both groups jumped up and reached levels of 80 - 90% after four years of AZT therapy. In other words, after four years most AIDS patients had died.

This extreme mortality eventually got noticed though, and accordingly the AZT doses were lowered around 1990, because it also became obvious that the bone marrow couldn't stand the chemotherapy. Still, any antiviral therapy has been and still is a lifelong therapy. Only this year, after numerous problems with side effects were reported also for the newer drugs (protease inhibitors), they publicly consider drug holidays (Nature, Lancet, 2000). Now they state everywhere (see Montagnier) that the new therapy works, because mortality of AIDS patients has clearly declined. This, however, is nothing but a euphemism for lower toxicity by dose reduction.

An increasingly critical attitude by patients themselves, who have witnessed the AZT disaster of the early 90s and extensive literature on the AZT problem have generated a more critical atmosphere toward the therapy. And yet, declining mortality of AIDS is still attributed to the better therapy, and declining mortality correlating with increased use of protease inhibitors is demonstrated in a time frame (Palella et al. NEJM). What you can't see in that time frame is the fact that mortality had already been distinctly declining since 1990/91, the time when therapists noticed that AZT in 1500 mg doses were not tolerable for their patients (bone marrow suppression). At that time, however, we had already treated a whole generation of AIDS patients into irreversible immune suppression.

This AZT catastrophe is the reason for the ineradicable belief that HIV is the cause of AIDS. Moreover, it has led to the habit to use "HIV" and "AIDS" as synonymous terms. Epidemiological predictions are based on this assumption that HIV is the same as AIDS, and in respect of all countries with such HIV-test explosions they predict that catastrophic AIDS epidemics will follow. For the president of South Africa, Mbeki, the discrepancy between what European and US newspapers write about his country (drastic population reduction) and what is actually happening in his country (doubling of the population within the past 30 years), was striking. Hence he refused to follow the general (American) AIDS-politics and instead, called the meeting of experts who had the task to examine whether or not HIV was actually the cause of AIDS.

Two things had particularly startled him: First the extensive literature on AZT and the damaging effects of this substance. Secondly a paper by Max Essex that was published in the "Journal of Infectious Diseases" and which describes a strong cross reaction of HIV tests with antigens, that can be found in the bacteria which cause tuberculosis and lepra. That means, nobody in Africa or elsewhere in the world knows whether a patient suffers from tuberculosis because he is HIV-positive, or whether he is HIV-positive because he suffers from tuberculosis.

Another problem of the AIDS epidemiology is the following: By now about 30 afflictions, all of which were known before, are being renamed to AIDS in the presence of a positive HIV-test. This also is not an increase of diseases of course --but just a redefinition. This circular definition HIV+/TB = AIDS and HIV-/TB = TB makes the correlation HIV-AIDS appear 100%. For example, a patient who suffers from TB and who is also HIV-positive is today considered an AIDS patient. A woman who suffers from cervical carcinoma is today considered an AIDS patient, and so is a patient with a lymphoma. If they have antibodies against HIV.

The virus-AIDS-hypothesis and the media alarm connected to it (12 cover stories alone by the

German magazine Der Spiegel) has caused the biggest medical catastrophe and human tragedy, by driving countless numbers of people into fear and despair, by causing suicides and iatrogenic deaths, and is still doing so.

Possibly the end of this is in sight, if Mbeki will be successful with his AIDS politics and will ban HIV-testing as well as antiviral medication in his country, and instead, will fight tuberculosis that is progressing in his country and poverty that is connected to it. Tuberculosis has always been a good indicator for the weal and woe of a society (see the frequency of TB in Germany after the two world wars, Statistisches Bundesamt Wiesbaden). Modern tuberculosis however is now, after the introduction of HIV-tests, called AIDS and is treated accordingly. In India they showed me patients who had tuberculosis and sold house and home, in order to get the cure (AZT) from the West.

Hepatitis C

With hepatitis C we see a similar phenomenon, although the iatrogenic measure is not as drastic as in the case of the HIV/AIDS hypothesis. Here one can only expect a temporary therapy with interferon and Ribavirin ™, however this therapy too produces many side effects, and as I will show, it's also superfluous.

The birth year of hepatitis C is 1987. The laboratory for this job was nothing less than the Chiron Corp., a biochemical company that by now makes billions in sales with Hepatitis C tests. At that time they injected blood from a patient with a Non-A/Non-B hepatitis into chimps. None of the animals developed hepatitis. Just around day # 14 after the infection they showed temporary increase of liver-enzymes (transaminase). The animals were slaughtered, and the liver tissue was examined. They didn't find a virus. Being in deep despair they then searched for the tiniest traces of a virus, and amplified a little piece of genetic information, that didn't seem to belong to the genetic code of the tissue, via PCR. They assumed that this piece of foreign RNA must be the genetic information of a before undiscovered virus. Whatever it was, the liver tissue contained it in hardly detectable quantities, but they were able to build an antibody against it.

This antibody bestowed us the hepatitis epidemic insofar, as test explosions are taking place again and HCV positive patients are now told they carry a virus that after a latency period of ca. 30 years will generate a liver cirrhosis. Most of the HCV positive patients, however, don't have any symptoms of illness. Some have slightly increased transaminase, and real liver damage is almost exclusively a problem of those patients who have consumed alcohol and drugs before. Here we see indeed a big overlap insofar, as almost 80% of the drug addicts are HCV positive. Now we have to answer the question again, does the virus damage the liver, or the drugs and the alcohol? The 30-year latency period would then be an euphemism for the toxic effects of drugs and alcohol that can lead to liver cirrhosis after 30 years.

While two or three years ago newspapers had headlines like "Hepatitis C - underestimated danger; HCV - unrecognised danger; HCV - the new big plague, "It comes quietly but powerfully". We nowadays read more often: "Danger of hepatitis overestimated?". Prof. Manns from Hannover, who initially was one of worse case predictors, is now saying that - based on the available studies and on a cost-benefit-risk estimation - therapy for Hepatitis C can be seen as a relative counter indication.

This new view when it comes to an estimation of hepatitis C has the following background: Last year Seef et. al published a big study in Annals. of Internal Medicine, that was carried out with GIs whose serums had been frozen 45 years ago. A follow-up over 45 years showed that there are practically no differences between liver diseases of HCV positive and of HCV negative people.
This indeed leads to the consideration that the risk of a HCV positive person developing liver cirrhosis later in life was apparently massively overestimated. And it makes the theory appear more plausible that liver toxic substances like alcohol and drugs, called "cofactors", are actually the main factors. Hence, a positive HCV test obviously has no clinical relevance. Accordingly, antiviral treatment for HCV positive patients doesn't make any sense.

Moreover, medical treatment of liver diseases has been considered paradoxical by leading hepatologists over many decades, because practically all substances damage the liver in one way or the other, because the liver is the main organ for metabolism of toxins. For example, Benuron, that is used during an interferon treatment one gram per day. Remember in this context the Fialuridine disaster - a treatment attempt - a few years ago, where a couple of patients died, and others could only be rescued by liver transplantation (Hoofnagle et. al).

A German scientist and his team were able to find the sequences named HCV in human DNA of healthy HCV negative individuals. So, it's imaginable that HCV positivity can be produced endogenously when liver cells get damaged by toxic substances like alcohol or drugs and then generate these sequences. This would explain the relatively strong correlation between HCV positivity and alcohol/drugs.

In the case of hepatitis C – its similar for hepatitis G - we can apparently still hope for a self-correction of science, because of the lack of clinical evidence. HCV positive liver cirrhoses occur almost exclusively in drug users or alcoholics, while a significant group of people who are HCV positive and develop a liver cirrhosis at the age of 50 and who are free of nutritive-toxic liver damages, does practically not exist.

Medical publications and the general press are promoting the epidemic-like character of the hepatitis C plague. Recently, in Itzehoe a HCV positive surgeon allegedly infected many of his patients. But one has to consider that prevalence of hepatitis C antibodies is relatively high in the population, so that it is easily possible that 2% react positively to HCV tests, that means 40 cases out of 2000 would match the general degree of "infection".

BSE (Bovine Spongiform Encephalopathy)

Now the atmosphere of plague fear culminates in the BSE hysteria --where we have not one case of illness in our country [Germany], and still you can read about the BSE crisis or BSE plague in all newspapers. Here again we see the phenomenon of a test explosion, insofar as the Swiss company Prionics has their BSE tests ready for the market and is distributing them. Here again a positive test case is equated with a case of disease. The plague atmosphere created by this is even supported by the panic which comes up with the hypothetical notion that mad cow disease can be transmitted to humans by them eating beef and will appear as the new variant of the Creutzfeld-Jakob disease. The media heat up this atmosphere of plague fear by dragging putative victims in front of the TV cameras although the disease is only diagnosable post mortem.

While all epidemiological data available so far contradict such a connection, this is still the big fear, which drives scientists and politician to the current totally overdone safety measures (mass slaughter of cows).

If we want to understand this fear, we must browse back a some years, and consider the work of Carleton Gajdusek. Gajdusek did research in Papua New Guinea in the 70s, on a kind of dementia, which was prevalent mainly in the female population there. The disease Kuru was observed as being endemic in two tribes whose members often married each other. These so called transmissible spongiform encephalopathies which Kuru belongs to, the Creutzfeld-Jakob disease, the familiar insomnia and the Gerstmann-Sträußler-Scheinker syndrome appear sporadically or genetically caused and of autosomal dominant origin. These diseases are fatal within 5 years. They are extremely rare, frequency is around 1 : 1000000 and within a family with a frequency of 1 : 50 --which is a good argument for a genetic cause.

But Gajdusek received the Nobel Prize for his concept of slow viruses and thereby established the transmissibility of those spongiform encephalopathies. However, if we observe his experiments he tried to prove the transmissibility with, we have to wonder today that the scientific community at that time accepted those papers as proof for transmissibility.

Neither the feeding of infected brain tissue nor the injection of it affected the lab chimps, only one bizarre experiment led to neurological symptoms in the chimps, and this was intra-cerebral inoculation experiment. On these experiments the transmissibility of those diseases is based! Hardly evidence for Gajdusek´s cannibalistic hypothesis, which postulates that the disease in humans could be caused by the consumption of, infected brain. Burdensomely we have to add that Gajdusek is the only witness alive for cannibalism in Papua New Guinea. One teams of anthropologists that examined the case, found stories about cannibalism but no authentic cases. So, about Gajdusek´s Nobel Prize we can only say, if his stories are not true, they have nicely been made up anyway. Despite these inconsistencies (intra-cerebral inoculation experiments) for proof of the oral transmission path the notion of oral transmission is now so established that we actually fear the consumption of beef. According to Gajdusek´s attempts, we would only have reason to fear something, if we made holes in our head and inoculated the infected brain of mad cows.

Also on the cannibalistic hypothesis the assumption is based that by feeding of infectious animal meal the plague got started. Because of the general acceptance of this hypothesis it is entirely neglected that the epidemiology of BSE does not match the feeding of animal meal at all. Great Britain for instance has exported tons of animal meal to the Middle East, South Africa and also to the USA. In none of these countries BSE occurred Instead, BSE cases almost always occur in Great Britain (99%), Switzerland and North Ireland.

One explanation is in the case of BSE again the intoxication hypothesis. 1985 in England a law came into force which forced British farmers to pour Phosmet ™ along the napes of their cows. Phosmet is an organphosphate that is used as insecticide against the warble fly. This substance was used in relatively high concentration only in Great Britain, North Ireland and Switzerland, and the law didn’t allow an exception. A British farmer, Mark Purdey, noticed that his cows from organic production didn’t develop BSE although they were fed by animal meal, but never treated with organphosphates.

The British Government knows this context, and in the early 90s the law was taken back, because a connection between the organophosphate and the occurrence of BSE was very likely. Organophosphates can change the alpha helix structure of proteins. According to this measure, BSE cases started to decline from 1993 on. Actually, the British inquiry committee admits that organophosphates are apparently a cofactor for BSE. Toxicologically it is known (Lüllmann, Kuschinski: Lehrbuch der Toxikologie) that chronic intoxications with organophosphates lead to "the clinical symptoms of polyneuropathy. The basis is axon swellings and fragmentation and eventually demyelinization of peripheral and central axons".

However, the BSE inquiry committee refuses to accept the organophosphates as the sole cause. But one question comes up. Why do not all those cows get the disease that was treated with organophosphates? Here we must consider: The dose makes the toxin - and even if all cows get the some quantity it depends on the diffusion distance whether the toxin reaches the central nervous system and can start its damaging activity.

Thereto the observation of British farmers: meagre milk cows are significantly more receptive to BSE than the fatter beef-cows. If one pictures the diffusion distance the nerve toxin takes after being poured over the nape of the cows, one can easily imagine that the thickness of the subcutaneous fatty layer is quite crucial for whether or not a cow will develop BSE. As lipophilic substances the organophosphates are buffered in the subcutaneous fatty layer.

Summary

But if a toxin can speed up the outbreak of a disease, like alcohol can contribute to liver diseases, then it can also be the sole cause. However, if Phosmet would be declared as cause of BSE, compensation lawsuits in billions would wait for both the British government and the manufacturer of the insecticide. This is certainly not desirable for them, so they prefer to surround the basically clear context in a fog of prions.

Intoxication hypotheses are easily testable and in contrast to the virus or prion hypotheses also falsifiable. They can be examined toxicologically and epidemiologically and then we can either accept or reject them.

For AIDS, the intoxication hypothesis would make following predictions:
All patients, who die young of AIDS, must have used recreational or antiviral drugs over a longer period. There must not be a significant number of people who die of AIDS at a young age and who are drug free and haven't taken any antivirals.

For hepatitis C it would mean, that there is no significant number of people who die of Hepatitis C caused liver cirrhosis in their midlives and who are drug free and alcohol free.

And for BSE the intoxication hypothesis would mean that only cows who have been treated with organophosphates, develop BSE, and inversely, if a significant number of cows with no organophosphate treatment would develop BSE, the intoxication hypothesis would be proven wrong. As elaborated above, epidemiological and toxicological data suggest that chronic intoxications are the real cause for the named diseases AIDS, HCV and BSE. Why these plausible hypothesis aren't investigated further, this is a topic one could write a book about which could have the title "conflicts of interests".

Infection hypotheses can help making billions of dollars:

1. The antibody business: Millions of screening tests are distributed, each blood sample needs to be tested (4 millions in Germany alone)

2. The therapy business: Antiviral medication, 3 or 4 or 5 fold combinations, AIDS can't be topped in this department.

3. Possibly vaccinations: Here, however, the concept of the new big plagues gets in the way of itself, because this has brought up the central paradox of immunology. Since the beginning of HIV they have told us: He who has antibodies to HIV, will die, instead of, he who has antibodies to HIV will live, which would meet our vaccination concepts. How many HIV antibody negative individuals would like to get vaccinated, in order to have antibodies to HIV afterwards?

With intoxication hypotheses on the other hand you cannot make any money at all. The simple message is to avoid the poison and you won't get sick. Such hypotheses are counterproductive insofar as the toxins (drugs, alcohol, pills, and phosmet) bring high revenues. The conflict of interests is not resolvable: What virologist who does directly profit millions from their patent rights of the HIV or HCV tests (Montagnier, Simon Wain-Hobsen, Robin Weiss, Robert Gallo) can risk to take even one look in the other direction.

What physician who has treated AIDS or hepatitis C patients over many years in good faith in the virus hypothesis and with high personal input, can look in the other direction? The more so as he must get the feeling, due to seemingly plausible changes of surrogate markers, that he is on the right track. Everywhere in the world children are treated according to this principle. Healthy children get antiviral therapies, in order to "delay the outbreak of illness", that is, a clinically healthy HIV pos. child gets a therapy, and any affection that appears under this therapy will be blamed on the "basic disease" or interpreted as therapy failure because of the virus developing resistance. In other words, the child has no chance to escape.

I have experienced myself --at a trial in Canada (I was ordered to as an expert of AZT), how healthy kids were taken away from their mother who had been HIV+ for 15 years and who was allowed to refuse anti-retroviral treatment for herself but not for her children.

Similar was a judicial sentence in England where a HIV positive couple refused to get their newborn tested. The judge said that the child must be tested, because in the case of a positive test result immediate therapy would be necessary.

Even study results that shed light on the AZT use of pregnant women aren't able to wake up the authors. They describe a 5 - 6-fold higher risk of a rapidly progredient course of HIV infection for those kids whose mothers have been treated with AZT during pregnancy, compared to children whose mothers have not got any AZT (J. of AIDS, 2000). At least our efforts in Africa on the panel seem to have somewhat impressed the Americans, because a few weeks ago the NIAID (National Institute for Allergic and Infectious Diseases) announced a big multi-centre study that included a therapy branch without antiviral therapy. So, after 13 years of aggressive long term therapy now a "U-turn" over to what until now has been considered not justifiable - a real placebo control with clinical endpoints, planned for four years.

It would be my wish that I have seeded at least a few doubts with my lecture, and I hope to inspire a broader discussion.

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