Throughout the 19th century, the medical authorities pro-
claimed for political reasons that cholera was not contagious. The reason for such strange behavior was that quarantine would stop trade with Asia and with Moscow, where most epidemics came from. Such was the case in the 1832 cholera epidemic when 100,000 people needlessly died in France, because no attempts were made to control the spread of the disease.

Today, for political reasons, the scientific community is complicit in the cover-up of the century in relation to AIDS. As with cholera in the 19th century, AIDS is said to be non-
contagious, for fear that “authoritarian measures” might be taken, and above all to save money. Hence, public health measures are not taken, the looting of Africa proceeds as usual, and promising scientific research areas are blocked. Allegations that we may be close to finding a treatment re-
mind one of the 19th-century physicians convinced that treating cholera patients with blood-sucking leeches improved their condition.

The October issue of Scientific American, the “reputable” organ of popularized scientific research, is entirely devoted to AIDS with the title: “What Science Knows about AIDS.”

Indeed, the list of contributors is a “who’s who” of the AIDS specialist’s club: Gallo, Mann, Haseltine, and others. The fraud starts with the first pages, when Robert Gallo and Luc Montagnier pretend to write something together on the discovery of the virus, as if it had been a collaborative affair, albeit unwittingly . . . . It goes thus: “One of us (Gallo) . . . did (etc.), to another one of us (Montagnier) . . . .” The result is queer, like the offspring of a sexual affair between a chicken and a fox, if you will forgive me the thought. No more need be said.

Ostensibly ten AIDS-related topics are examined in Scientific American, from the “probable origin” and cellular activity of human immunodeficiency virus (HIV), on to treatment and vaccines being looked at today, or the epidemiology and “social dimension” of the disease.

Methodologically the worst articles are those on treatment and vaccine research, because the wealth of “facts” is only matched by the poverty of ideas and the overall fraudulent claim that “we are well on our way” to solutions to the problem.

Dubious therapies

“AIDS Therapies,” authored by Robert Yarchoan, Hiroaki Mitsuya and Samuel Broder (from the Clinical Oncology Program of the National Cancer Institute—NCI), is a case in point. The authors are among the people who initiated the use of AZT (also called azidothymidine or zidovudine) in the treatment of AIDS patients, a drug which they rediscovered from the work done on mouse retroviruses (so they write) by people at Max Planck Institute, the Belgian Rega Institute, and others. The effort was financed by Wellcome Research Laboratories which must have made a handsome profit since the work started.

As everybody knows, AZT is the antiviral drug most widely used on AIDS or pre-AIDS patients today. The authors pretend that AZT offers a solution, albeit a temporary one, in that it slows the progression of the disease, and early administration could be successful in thwarting the development of full-blown AIDS. They add, “The drug can be toxic, particularly to bone marrow, so that patients on AZT often develop anemia [a decrease in red blood cells] and in some instances low numbers of white blood cells and platelets as well.”

This a gross understatement, or rather only half the pic-
ture, since AZT also attacks nerve fibers rather rapidly. Under such conditions, can it be asserted that AZT is a "solution," or even the beginning of a solution worth gloating about? They go so far as to excuse the toxicity of AZT by saying that "penicillin too is toxic"! AZT does not clear the organism of the HIV virus from the organism, it does not even prevent the formation of syncytia (clumping together of cells, which is one of the most characteristic actions of the AIDS virus), and it is highly toxic. It cannot be administered for a long time without dramatic side effects, and it has not saved one life. How this drug can be compared with penicillin, which saved millions of lives, is beyond imagination.

Another "solution" coming on the market today is soluble CD4, "rCD4" as it is called. The idea is that the rCD4 will bind to the glycoprotein gp120 of the HIV virus, and thus prevent it from binding to the CD4 molecule of the lymphocytes. There are also several problems with that approach, which is going into the first clinical trials at present. First, it could be toxic to a certain type of naturally produced antibodies, known as MHC class II immunoglobulins. Thus it could produce its own form of immune dysfunction resulting in clinical problems similar to AIDS.

Beyond that, the evidence is slim that HIV only binds to the CD4 molecule, as non-CD4-bearing cells can be infected with the virus. Obviously, there are other binding mechanisms which we do not, as yet, understand.

Anything that fails to get rid of the intruder altogether, while being toxic to the host is going to fail in the long term. This is precisely the same problem we face in cancer chemotherapy. So far, unfortunately, with all the efforts which the authors mention, and they seem to be only interested in pharmaceutical companies' efforts, not one patient has survived the disease, or even survived longer without side effects.

While pretending to be exhaustive, the article is mostly oriented toward Anglo-American laboratories. For instance, it fails to mention the immune stimulator "immuthiol" developed by a French group, which has the merit of having some beneficial effect for cancer patients and of being non-toxic; or the effort by some Japanese to develop interferons that also have some beneficial effect and are non-toxic as well. But the main problem is in the extremely reductionist approach to the subject:

The authors break down the activities of the virus into a step-by-step factory of sorts, and suggest that if we know each and every individual part of the time sequence in the virus activity, we could successfully intervene at any point in the process.

The list goes as seen in the picture: "Ways to intervene." What is missed is twofold: 1) How can we distinguish the virus from the host cells? That is the first useful question to ask ourselves. All the more so, since the creation of antibodies seems to be part of the problem in an auto-immune-type mechanism. The second is a question that disagreeably tickles all the hairy molecular biologists: what about the extracellular activity of the virus? What Montagnier calls the "mysterious" "action at a distance"? Neither he, nor all the people mentioned in this article in the search for treatment even looks into the question.

It is reported that some researchers are creating "anti-idiotypic antibodies," antibodies to antibodies of CD4, which would presumably get at the gp 120 surface molecule of the virus, but the problem is the reaction of other cells, such as macrophages, to such antibodies. Just as with "rCD4" we are merely apprentice sorcerers in immunology, who don't know the consequences of our intervention, as all honest specialists will admit.

The article on "AIDS Vaccines" is by Thomas Matthews and Dani P. Bolognesi (from the surgical virology laboratory at the Duke University Medical Center).

They write, "A rich tradition of vaccine research guides the efforts to develop an AIDS vaccine." True enough, but no vaccine has even been found against a retrovirus, which the authors admit in passing, without stressing the point, to say the least. But they do add a truthful and most important remark:

"The fact that HIV attacks the cells that are responsible for defeating infection adds its own twist to vaccine development. In particular some investigators are concerned that a vaccine could actually enhance the infection of the virus. Certain cells of the immune system have receptors that bind
to antibodies opposite the antigen-binding region. Macrophages are among these cells, and macrophages are a target of HIV infection. Antibodies attached to free virus could therefore be attracted to macrophages, increasing the chances that a macrophage will become infected. Hence raising antibodies to HIV by means of a vaccine could conceivably facilitate rather than deter the spread of the virus [emphasis added]."

That is the main reason why no vaccine has even been found against Visna or other animal retroviruses. As French researchers have said: the problem lies in the activity of the macrophages that harbor, carry, and produce HIV. Besides, the macrophages are thought to be the main vehicle bringing the HIV virus to the brain.

The details supplied as to who is doing what in the field are not very relevant, since the approach remains traditional, and so partakes more of a random experiment than anything else. Today one wishes more medical researchers would think like the French Pasteurian Charles Nicolle who confided to his pupils in 1937, "I hate random experiments, I love experiments to think."

In the French medical community generally today, I have found more and more top people who say that a vaccine in the traditional sense of the term is out of the question. Those researchers who have been best trained in Pasteur's method of vaccination do not believe it will or could apply in the case of AIDS; no linear extrapolation will do.

One of the additional problems, we could say, is that beyond going to the "head" of those normally in charge of defense, the lymphocytes and the macrophages, the virus also mutates, and mutates faster than anything that has ever affected mankind so far.

In birds, retroviruses effect one mutation per replication cycle. Say HIV would do that in fact, this does not mean that it would be so different as to be unrecognizable each time, of course, but we do know today (as demonstrated by several people during last June's WHO AIDS conference in Stockholm) that HIV mutates very fast, from one person to the next, during the course of infection in the same person (for example, virus retrieved during the beginning phase of the infection is much less active and much less cytopathic than virus retrieved later in the course of infection.) In addition, virus obtained from the cerebrospinal fluid and from the peripheral blood of one single patient has as different biological activity as two different strains.

"The Origins of the AIDS Virus" by Max Essex and Phyllis Kanki (Harvard School of Public Health) is so banal and vague, it reads like a fairy tale. It says that SAIDS (Simian AIDS) only infected captive Asian monkeys (not those in the wild) and that African Green monkeys were often found to be infected with SIV but did not develop SAIDS. It says chimpanzees can be infected with HIV but do not catch the disease, and that the chimps might have been exposed to a close relative of HIV and developed resistance. This information, and what is inferred from it, is just nonsense, since the animals do get clinical manifestations after infection with HIV, as I am informed of that fact. So the article is far from representing the state of research today, or even that of a year ago!

As for the assumption that HIV-2, which is prevalent in Western Africa (as opposed to HIV-1 in the eastern part) is less pathogenic than HIV-1, that too, I would say is disinformation: Several people have died of HIV-2 induced AIDS in European hospitals already, and, as with HIV-1, the neurological manifestations can be impressive.

The possibility that AIDS began with a laboratory accident is, of course, ignored by the authors, who stick to the monkey story, in effect, saying we know nothing about the origin of AIDS.

An "unprecedented threat"

"The International Epidemic of AIDS" (Jonathan Mann, Piot and Chin) admits: AIDS represents an "unprecedented threat to global health." WHO estimates that 250,000 AIDS cases have already occurred and that 5 to 10 million are HIV infected today. Bad as those figures may sound, they are still far from the truth as the number of infected in the U. S. alone is 4 to 5 million today. The authors admit to the obvious correlation between genital or anal lesions and transmission in the homosexual community, and that areas of high HIV prevalence in Africa are also the areas of prevalence for sexually transmitted diseases (STDs) such as syphilis. But they use that to promulgate the STD theory of epidemiology, saying that there is no evidence of transmission by "food or water, biting insects, coughing, or sneezing . . . no casual transmission."

And the maps of the world accompanying the article tend to indicate a "homosexual and IV drug user transmission only" in America and Western Europe, and special heterosexual transmission in Africa (they admit to up to 20% or even 25% of people infected in areas of Central Africa). This is another hoax, which the Masters and Johnson report on heterosexual transmission in the U. S. should have dispelled. Presumably, this is what prompted Robert Gallo at the most recent international conference on AIDS in Africa, which took place in Tanzania this September, to suggest that only Africans could be used to test vaccines, because there were only "special populations" affected in the United States. This guinea pig proposition provoked a protest by most of the African health ministers present.

Mann, et al. add that, "The Harvard Institute of International Development estimates that, by 1995, annual loss to Zaire from AIDS deaths will be $350 million, 8% of GNP, or more than all the international yearly financial assistance received from the West . . . Economic loss to Central Africa would reach $980 million." Nonetheless the authors end by congratulating themselves and WHO: "There is no precedent in history for public health effort for the speed, intensity or scope of the global AIDS effort." (To spread condoms?)

From Walter Reed Institute, Robert Redfield raised the
interesting point that it is wrong to ask physicians to explain AIDS, because AIDS is just one particular late manifestation of the HIV infection, which is what one should ask about. The problem is that, while arguing as to the advantages of early tracking of the HIV infected (a strong argument in favor of mandatory testing), only the immunological effects of HIV are taken into consideration in the Walter Reed classification (a more intelligent one than the Centers for Disease Control for sure) which evaluates the progressive deterioration of the immunological system.

Redfield notes that the deterioration in lymphocytes means that infections with mycobacteria, viruses, fungi, and parasites, will develop more commonly than infections by bacteria. This is because bacteria are not repelled by T-cells, but instead evoke an antibody response from the B-cell system, the other branch of the immune system. This is important to understand the facility with which the mycobacteria, such as tuberculosis, develop in AIDS patients.

In the author’s evaluation, the virus proliferation evolves along with the virus toxicity, which means that the person is more contagious at the onset of the disease, before there is any significant immunological response, and then again toward the later phase of the disease. “More virus in the body means greater infectivity,” obviously! And he makes a strong point: Early diagnosis of HIV infection, by more screening, means that we can follow the evolution of the immune system better, and have a more precise evaluation of the contagiousness of the person. We can only regret that Redfield seems to push aside the neurological manifestations of HIV, which represent a clinical picture of the utmost interest in developing a grasp of the disease. He even says that neurological manifestations could be just opportunistic infections (e.g., not due to HIV).

What is remarkable about this type of media coverage, is that it seems to have the double intent of dumbfounding the public, and, more importantly, of getting some decent people to degrade themselves into writing half-truths, such as Montagnier, who is normally not afraid to say that he alone discovered the virus. We are face to face with that Inquisition which Professor Benveniste denounced for the behavior of Nature magazine toward his research (See EIR, Vol. 15, No. 34, Aug. 26, 1988, page 20). This type of coverage serves as a message to the scientific community as to the “acceptable” ways in which to shape their thinking and research, lest they be left out and get no funding. It is a sort of guarantee as to “all that which science shall never know about AIDS.”

But the extent of the epidemic, the grim future, the incredible costs, and the threat to a whole continent, are recognized even while appropriate action is blocked.

The social dimension

“The Social Dimension of AIDS” by Harvey Fineberg (Dean of the Harvard School of Public Health), has some hairy news to tell:

- Infection among drug users is markedly on the rise.
- The cost to the U.S. economy and health care system will be horrendous: “The U.S. Public Health Service recently predicted that 450,000 cases will have been diagnosed by the end of 1993, extending the 270,000 by the end of 1991. Personal medical costs for AIDS patients during 1991 have been projected to reach levels of between $4.5 and $8.5 billion. Other costs are subtler,” Fineberg continues. “When a hospital adopts universal precautions requiring frequent use of disposable gloves, gowns, masks and protective eyewear, hires additional infectious disease specialists and infection control personnel, follows special blood-screening and laboratory procedures . . . such costs are spread over all patients and are not found on those having a diagnosis of AIDS.”
- Fineberg informs us that in the United States, “One in five AIDS patients has no insurance, 40% are covered by Medicaid, (more than four times the proportion in the general population).” This indicates that AIDS affects especially the black and poor population in the U.S. generally.

“Medicaid only covers 40% of those with income below the poverty line, and frequently pays less than the cost of care.” (The American Medical Association has submitted a bill to Congress to extend Medicaid to the 20 million or so people not yet covered by insurance who are below the poverty line; it would double that budget.)

The problem of rising epidemics of opportunistic diseases such as TB is also raised:

- “HIV can also indirectly contribute to the rise of other infections in the community. After declining for many decades tuberculosis has began to increase in the U.S. Between 1984 and 1986 reported cases jumped 36% in New York City. Today, these new cases are found mainly among patients with HIV infection, but as more people in the community develop active tuberculosis the risk of spread to those not infected with HIV will increase.”

The Fusion Energy Foundation was first to point out this HIV-TB interaction, seen by tropical disease specialists in Florida and Haiti but denied by the CDC, until the recent period, notably since the Stockholm Fourth International Conference on AIDS last June (see report in EIR, Vol. 15, Nos. 26 and 27).

In late October of this year, the French virologist Professor Chrétien from St. Louis Hospital reported his finding that TB which was decreasing in France by 11% per year until 1984; it subsequently decreased by 8%, then only 1.3% in 1987, and increased by 2% for the first half of 1988 (compared to first half of 1987). He pointed out that HIV infection was activating TB, and that there had been “laxity” in applying TB control measures (which include quarantine) and that these ought to be revived along with HIV control measures. About the “social dimension of AIDS” in the Third World, Fineberg again doesn’t offer any solution, but he does write something about the stark reality of the matter: “Demographic projections suggest that the long-term impact of AIDS on those populations [Africa and the Caribbean] may be similar to a prolonged war.”