

On the nature of AIDS. New hypothesis.

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Abstract

Thirty years have passed since the AIDS epidemic began. It is possible to draw some conclusions: vaccines and treatment methods have not been developed, and the viral cause of disease has not been scientifically proven. The time has come to consider a cancer hypothesis. Previously, it was not discussed only because leukemia is considered a disease of immature cells. In 2006 it was confirmed by several methods, that Hairy cell leukemia is leukemia of mature B-lymphocytes. In 2008 a comparative analysis of the resistance of immature and mature blood cells revealed the high resistance of the latter. This property can explain the later onset of mature B and Th (T-helper cell) leukemia in the repertoire of leukemias. The leukemia hypothesis gives answers to the questions like why there are more virus carriers among females, whereas there are more males among the ill and other questions. If Th cells became immortal cancer cells, it is contrary to their functional purpose in the body, i.e. it launches an immune response that causes its own destruction. In humans, there are mechanisms that destroy cells which have stopped functioning. The disease lasts from 2 to 5 years; during this time normal Th cells are replaced by cancer cells and immune deficiency leads to patient's death. During the 30 years one man only has been saved and healed by means of a bone marrow transplant - the common treatment of leukemia. This successful treatment should be the best confirmation of the hypothesis. The coincidence of pre-AIDS symptoms and pre-cancer symptoms with Chronic Fatigue Syndrome (surveyed more than 250 sources and own observations) lead us to think that there is a single mechanism causing these diseases. The Leukemia and Lymphoma Society reminds us to: "Escape chronic exposure to certain chemicals". Long-term exposure of humans to small amounts of chemicals from non-industrial sources of room air contamination is accompanied by release of interleukins, changes in peripheral blood Ph, and loss of DNA structure in lymphocytes. Defense reaction of cellular immunity manifests many unhealthy symptoms as well as fever.

In the early 1970s new diseases associated with activation of non-infectious immunity were predicted. It was also predicted that the virologists who were barely familiar with non-infectious immunity would make the connection between viruses and new diseases [42, 50].

Ecologists wrote that the vast majority of books themed "environment and human" focused on humans harming the environment, but not vice versa [30,64]. People's health is defined by environmental conditions just as much as by inherent factors [6,48, 51]. Long-term human exposure to small amounts of chemicals issued by non-industrial sources of room air contamination was noted [5, 59]. The Massachusetts Department of Environmental Protection created a work group known as "Indoor Air". This group cooperated with Institute of Scientific Research of Human Ecology and Hygiene of the Environment in Moscow, Russia directed by G. Sidorenko, which conducted research in apartment buildings equipped with gas or electric stoves and in offices of administrative buildings, where synthetic construction materials were present [22, 23, 24,66]. The most informative research was conducted not in the laboratory, but in cooperation with the Moscow Sanitary Epidemiological Station (SES), which was triggered by complaints of ailments and indoor odors. Those were quite comparable with full-scale studies in cases that met all requirements for such studies.

A mysterious disease occurred in 1982 in workers at the Ministry of Energy information center. 120 out of 125 workers felt extreme fatigue and became odor sensitive some months after the building was remodeled. The other five employees who worked mostly outside the building presented no complaints. All

the sufferers were young software programmers who worked in the building for a number of years; 120 workers had thought they were ill at the beginning of our study. Each of them asked for medical assistance not less than eight to ten times with complains of extreme fatigue which was not correlated to physical load and did not disappear after continuous rest. They experienced muscle and joint pain, tumescent and painful lymph glands, sore throat and fever. All three pregnant women lost their babies. All of the sufferers noted discrepancy between their health and medical diagnoses, such as acute respiratory disease and depression. General analysis of the air with a mass spectrometer and air samples of separate components showed that formaldehyde emitted by the particle boards used in wall construction was the major air contamination source. The formaldehyde concentration in the room air varied from 0.7 to 1.5 ppm within a year after they were installed. Surveys showed inexplicable depression and multiple complaints in all the sufferers, such as: "I feel as if I've been run over by a truck – my whole body aches, I don't want to live, I'm not interested in anything, and I'm like a walking corpse." When examined, objective symptoms of the disease in sufferers were extremely limited: swollen lymph glands, edematous soft palate, moderate fever, characteristic change in voice pitch.

Similar complaints were presented by people living in other buildings with inner sources of chemical air contamination. In some cases, the reason was the scouring cloth saturated with formaldehyde that construction workers used for filling in pinholes in defective wall panels. In others, the reason was roofing material containing phthalates. Literature sources state, that long-term exposure to formaldehyde and phthalates results in such immune status changes as antibody stimulation, abnormally high concentrations of circulating immune complexes (CICs), and other abnormalities [16,32,40,49]. Investigation was conducted after SES received numerous complaints from people living in multi-story apartment buildings. Phthalates were emitted by synthetic material used for roof repair within the range of allergenic action concentration. The disease occurred in all the people living on the top floor in the five to six months after the beginning of the repair and resembled influenza. However, specific peculiarities became apparent later and included: paroxysmal origin of the disease; short duration of the bout (often not longer then a day); specific pharyngeal edema; change in voice pitch not connected with hoarseness or appearance of overtones; increase, not loss of appetite, probably because victims felt better while eating; strengthening of a sensation of weariness at bed rest, local cold sweating at night, local recurrent pus rash, neurological symptoms. Later it was noticed that the disease symptoms in the sufferers resembled pre-cancer and pre-AIDS and had the same description as Chronic Fatigue Syndrome (CFS), a disease registered in 1988 [27]. Understanding the reasons for such a coincidence is crucial in ascertaining the mechanism of cancer and AIDS development, which is currently unknown (Chart 1).

Analysis of patient cases of those registered in the All-Union Allergic Center in 1984 was conducted to identify the importance of non-industrial air contamination sources [14]. It was discovered that for the most part, sensitization was not connected with the employment of the affected people in the production and use of chemical agents. Workers constituted only 15.4% of the more than 900 observed; other population groups (employees, students and retirees) constituted 84.6%, which corresponds to WHO data which announced that 82% of the carcinogens (being chemical allergens at the same time) are present where we live. Our investigation at a *tire factory* showed that workers found it easy to tell the cause of ailment at the enterprise where sources of contamination agents are well-known. At such an enterprise the rate of employee turnover is extremely high.

The practical goal of our research – to define a diagnosis in sufferers in office and residential buildings – made us turn to the works of immunologist *F. Dixon* (US) who in 1959 described Immune Complex Disease (ICD) by intravenous injection of antigen in animals for some months [6]. Research results conducted in the field of space medicine clearly showed that not less than 90% of formaldehyde and butyl phthalate in inhaled air enters the sufferer's body via blood and causes an anaphylactic reaction [10, 34]. ICD is not fatal like anaphylactic shock, but remains rather serious [18, 24, 38]. It was noticed in our research that the highest disease levels in employees corresponded to the highest levels of room air contamination with a delay of seven days, i.e. they corresponded to the maximum of Circulating Immune Complexes (CIC). When treating diseases related to immune complexes, immunologists paid foremost attention to antigen

removal [32, 42]. Therefore the most effective measure was to move the sufferers from the Data processing center to another building (in this case – removal of the employees from the antigen source). In residential buildings, the antigen was removed where possible.

Specific symptoms of CFS were described by doctors from the USA, Germany, and Australia. These included deep fatigue, low fever, muscle discomfort or muscle pain, sore throat, painful cervical and auxiliary glands, headache, concentration weakening and depression. All the CFS cases were registered in areas of environmental concern. The following faulty immune regulations were observed: increased level of antibodies (AB) and CIC. The disease was long-lasting. Versions regarding CFS etiology differed from psychic stress to viral infection [27]. The majority was inclined to think the disease had viral origin in spite of the fact that causation with none of known human viruses was confirmed by serological tests. Symptoms in those suffering in the Data Processing Center and the residential buildings were completely the same as CFS symptoms (Chart 1). It was concluded that CFS pathogenesis is defined by a normal cell-mediated immune reaction course when non-biological agents enter body.

Data from our research within the city, in different areas with different air contamination indices showed that the sensitization of people related to the air contamination level in the living area [14]. Due to the close relationship between antitoxic and antiviral immunity and its low specificity, sensitization spirals in the run of the disease. Multivalent reaction in those registered in the Allergenic Center was noted in 73% of cases. This means positive immune responses were not to one allergen but to multiple allergens (two to six) [15]. The viral CFS theory remained without proof. Some doctors decided to try treatment with an injection of immunoglobulin G (*IgG*) into the blood of patients without having discovered the mechanism of the disease appearance and on the basis of erroneous information on immunoglobulin G (*IgG*) level decrease. In an editorial article in *Lancet* magazine (1991, issue 337) titled "Ways that lead to death", the method of intravenous injection of *IgG* was severely criticized. In 82% of the cases using this treatment method, the patients developed complications lead to their death.

With the appearance of AIDS, two groups emerged-, those who believed in the infectious nature of the disease and those who believed that the cause was non-specific body sensitivity⁵ [7,25,29] . Therefore the search was conducted in two directions:

J. Sonnabend, S. Within, D. Purtillo and others believed that no specific factor existed and the disease occurred as a result of impact of multiple environmental factors that were connected with CIC formation. Followers of this position (19 scientists) formed the editorial board of *AIDS Research* magazine that was established in New York in 1983 [52]. Virology studies were conducted in The Pasteur Institute in France under the supervision of *L. Montagnier*, who was recognized as the *LAV* (*lymphadenopathy associated virus*) discoverer but all efforts to find the author's publications on the results of the immune deficiency virus in "*Index Medicus*" were fruitless. According to the materials of a conference it is known that *L. Montagnier* aroused indignation in his colleagues when he considered a virus as a sensitization factor and not an infectious one [35]. *R. Gallo* et al.(1984) managed to find *HTLV* (*human T-leukemia/lymphoma virus*), Type I in 4 out of 36 patients with full-blown AIDS [43]. For undefined reasons, he didn't consider the leukemic nature of AIDS despite common statistics characteristic of malignant tumors (according to statistical data, viral cancer occurs in 15% of cases, chemical cancer – in 85%). Although *R. Gallo* called *HTLV* a leukemic virus, he denied the leukemic theory of AIDS, since he didn't manage to find *HTLV* in all 36 patients with full-blown AIDS. Gallo may not have taken into consideration some well-known facts:

1. With viral cancer, as a rule, the full virus isn't evident. Only its markers in the genome are found [65]. Although Gallo didn't find signs of a viral presence in other patients, another 32 disease cases were AIDS as well, i.e. leukemia of a chemical origin.
2. No virus takes part in chemical carcinogenesis.

Attempts of all kinds to present AIDS as an infection continued. The *LAV* virus extracted from lymph node cells in patients with lymphadenopathy syndrome and lymphotropic virus *HTLV-III* were united into one – HIV (human immunodeficiency virus) – on the ground that both viruses are lymphotropic and exanthropic,

and have common heterogeneous antigens and damage *Tx* after continuous latency. HIV was made responsible for AIDS.

Results of a serologic survey on more than 500,000 people in the US showed that in an area with a high index of the detection of antibodies to HIV – 32 of 10,000 people – disease incidence was three times lower than in the area with lower index – 8 of 10,000 people. The **number** of women was greater among the HIV-positive, but among AIDS patients they were 10 to 20 times fewer than men in different age groups. According to statistical data from Center for Disease Control (USA), AIDS incidence in the Army (hundreds of thousands who went through mandatory preliminary testing for HIV) did not depend on whether the test was positive or negative. Disease incidence in medical personnel (more than 400,000 people working with AIDS patients) is no higher than the average incidence throughout the country. Clusters of leukemia, lymphoma, etc. were known before AIDS appeared, moreover it was noted this characteristic of the appearance of the disease does not mean the disease is infectious. It was discovered (Gamaleya Institute of Scientific Research of Epidemiology and Microbiology), that clusters of disease in children with pneumocystic pneumonia in orphan asylums was mostly defined by similar living conditions, rather than by presence of the opportunistic causative agent *Pneumocystic carinii* [58].

Chart 1

Pre-AIDS Symptoms	CFS Symptoms (Chronic Fatigue Syndrome)
1. Fever with temperature up to 38°C	1. Moderate fever. Paroxysmal disease nature
2. Abrupt decrease in physical activity	2. Fatigue that continues after rest in bed
3. Night sweats	3. Localized night sweats
4. Cough, labored breathing	4. Sore throat, edematous soft palate
5. Weeping dermatitis	5. Change in voice during episode
6. Lymphadenopathy	6. Tender lymph glands
7. Neuropathy.	7. Inability to focus attention; depression; memory impairment.
8. Muscle and joint pain	8. Muscle discomfort. Joint pain.
9. Clinically observed processes, which doctors considered autoimmune	9. Clinical symptoms connected to the mounting immune complexes[32]
Review of VNIIMI materials (230), 1986 [67]	Review of 26 sources and Government full-sized studies; GIS, 1995 [27]

Opportunistic virus (*passenger virus*), which is inactivated with UV rays affects cell culture in a way a live virus does; therefore it can only be an additional sensitization factor. When thinking about sensitization, one must also have in mind antibodies, sensitizing leukocytes, and the transfer factor (TF). TF sensitization activity is thousands of times higher than that of HIV [10]. TF is active in 0.0001 ml of blood and that is precisely why children from Elista (Russia), who received interferon injections with the same syringe, but different needles, got AIDS. Introduction of serum containing antibodies into a live body equals its sensitization with an antigen. Antitoxic antibodies can be transmitted from an immune mother to fetus prenatally through placenta, and after birth through the intestine with colostrum and milk [52].

Medical science has achieved great success in fighting infectious diseases. So why is it that there is no success in treating and preventing AIDS in the 21st century? [26]. Many virologists and immunologists were not prepared to understand the current situation. New immunology required major reconstruction of experts' way of thinking; previously major part of those experts had been microbiologists who fed on categories of infectious immunology." [50]

Altered immunity is a risk factor for cancer [1, 13, 28, 56, and 60]. If an infectious hypothesis turns out unproductive, it must be replaced with the one that will leave fewer uncertainties and allow making connections with a maximum number of facts characteristic of the disease.

It is a false belief that AIDS can be treated as infectious disease only because it is epidemically distributed. Many chronic diseases are widespread but it does not mean they must be treated as infectious diseases [58]. Viruses *LAV* and *HTLV* – III, marked as HIV, are opportunistic. Therefore they had to be accredited with such characteristics as changeability and special aggressiveness, which is not common in these viruses, and which were not proven when checked experimentally. It is not possible to artificially enhance the virulence of the virus [19].

Acquired Immunodeficiency Syndrome (AIDS) was introduced in 1981. Several reasons for the appearance of the syndrome were considered during a 30-year period:

- 1) toxic action of chemical agents, including medicines [7,8];
- 2) exhaustion of immune system as a result of its overuse in environmentally unfriendly conditions, because system of T-helpers protects body not only from viruses but from chemical agents as well [26];
- 3) contamination with retroviruses, that turned from opportunistic into deadly ones as a result of mutations.

-None of these reasons for disease were scientifically proved.

Now we can summarize all of the facts and leave less probable assumptions in favor of those more probable:

- 1) AIDS is not an intoxication, because by no means that everyone who died from AIDS used drugs, nitrite inhalers, or *AZT*.
- 2) Immune system is able to recover after a disease like no other body system, however, this was not the case in AIDS patients;
- 3) One of the steadiest viral characteristics is virulence. This was discovered in experiments by scientists, particularly by those who developed biological weapons. Therefore, they totally rejected the assumption that HIV was a virus that came from a test tube.

Australian scientists answered the question regarding how a non-deadly virus becomes 100% deadly. When creating the mouse contraceptive by introducing IL-4 gene into murine variola virus genome, they created a "factory" of excess interleukin-4 production. Immunity discordance led to the death of 100% of mice from the non-deadly virus. Thus appeared an assumption that in natural conditions, excess interleukin production can occur when there is a tumor involving T helper lymphocytes; 100% death of the infected happens not during two or three weeks, but during two to five years since the tumor develops gradually in comparison with the infection [19].

AIDS has different clinical presentations. A major and unifying characteristic is abnormal T-cell immunity, which successively performs multiple tasks: neutralizes antigen, saves it to memory, additionally sensitizes T-helper lymphocytes to produce a cascading immune reaction during a secondary response and finally makes a T-cell resistant to carcinogen action. Characteristics of tumor cells during chemical and direct viral carcinogenesis are similar, since in both cases a cell undergoes recombination; and they are different since in the first case there is a vector from its own source and in the other case the vector is viral. The viral and genetic (or *recombinant* in modern language) concept was introduced by L. Zilber in 1945. The concept was then experimentally proven and supported in 1960s by G. Abelev (1962), O. Sjogren (1963), H. Sjogren (1964), and others. According to this concept an oncogenic virus transforms a normal cell into a tumor cell hereditarily.

The cancerous nature of AIDS was suggested in 1990 (60), but it was not accepted, as leukemia was considered a disease of immature cells only. The malignant blood disease *Hairy cell leukemia (HCL)* was already known at that time. B-lymphocytes looked mature, but only the use of immunochemical and chemical-lighting methods allowed researchers to definitely acknowledging the existence of mature B-cell leukemia [4, 47, 62]. **Five times more men suffer from HCL than women. AIDS has the same ratio.** This could be one more piece of evidence that AIDS is not an infection, but leukemia.

In 2008, a comparison of the resistance of mature and immature cells to the influence of oncogenes was conducted. Mature lymphocytes appeared more resistant to the influence than immature lymphocytes (39). These characteristics explained the appearance of leukemia of mature B-cells (*HCL*) and aplastic leukemia of mature T-cells (*AIDS*) in the leukemic repertoire. AIDS specialists, who discovered HIV markers in *T helper* genome without full virus formation, unwittingly proved the correctness of the cancerous and not the infectious hypothesis. Presence of viral fragments in the cell-specific gene with no formation of the full virus is indicative of cancerous rebirth of the cell and not a sign of infection. This was proven and confirmed by eleven scientists from different countries (L. Zilber, 1945; M. Vogt, R. Dubecco, 1962; St. Subin, M. Koch, 1963; R. Huebner, W. Rowe, H. Terner, W. Lane, 1963; I. Irlin, 1965; V. Agol, 1990). Virologists completely ignored the above information and made further use of it as a prime consideration in favor of the infectious nature of AIDS. Along with the same disease manifestations, the absence of viral markers in the genome does not eliminate AIDS, but only gives evidence that leukemia has a chemical origin. This is true in 85% of cases (and more).

Based on their effect, chemical carcinogens are divided into non-genotoxic, including epigenetic mechanisms, and genotoxic, that cause irreversible changes in the genome. Thereby two carcinogenic strikes are considered [1,12,61]. The second strike, connected with genotoxic influence of chemical agents, was mostly studied, since the influence follows the rule:

dose (concentration) – time – effect

This rule can be objectively considered, and long-term effects can be forecast with the help of different test systems.

The first strike of carcinogenesis is non-genotoxic stress; this is a stage when physiological constants changing phenomena are accumulating. Some months of this stress are enough for Immune Deposit Disease (IDD) to develop as a result of cell-mediated defense reaction [3, 17, 37, 56, and 57].

IDD proceeds by way of anaphylactic attacks. Attacks are less strong than anaphylactic shock when active chemical agents enter blood with inhaled air, since they have relevantly weaker immunogenic characteristics, compared to foreign protein. However, a great number of biologically active agents are effused in the course of an anaphylactic reaction, which can be detected by laboratory tests and by the clinical presentation. Thus, it becomes possible to fill a rather blurred definition of *non-genotoxic stress* with quite specific content, namely with **laboratory data and clinical signs of CFS, pre-cancer, and pre-AIDS**.

The second carcinogenic strike – genotoxic stress – is connected with the destabilization of the genome structure. The destabilization indicator is used in ecological studies to estimate the degree of chemical load in the organism [21]. One can assume that tumor formation is preceded by genome rupture in one of the growth regions. There can be no reparation for genome rupture in the growth region upon exhaustion of the cell's energy resources. Therefore, the cell protection program is forced to change. In this case, reparation process is substituted by “reparative synthesis” with the insertion of nucleotide, which appears in cell immune protection system, i.e. in the source that is not connected with genome. (20, 31, 46, 61, 62, and 63).

The World science has received sufficient facts to allow consideration of the entire leukemia development process, starting with the very initial, preliminary, immune system response to a chemical substance, and ending with the appearance of Th leukemia (44). Substance with two chemically active centers is potentially hazardous, since it can bond to RNA and protein [24]. A natural adjuvant, which has this composition is known as “Lawrence’s transfer factor” (TF) [34].

A substance that is bound to TF₂ undergoes neutralization in macrophages that produce anti-RNA. Long double-stranded RNA molecule is created from RNA and anti-RNA as a result of a complex process in macrophages [38]. Its short section consisting of 20 to 24 nucleotides, known as RNA-interference (RNAi) complex, is able to interrupt protein synthesis in the cell [9]. After inhibiting protein synthesis, RNAi becomes sensitive to Slicer enzyme influence, which divides it into two strands. Anti-RNA with antigen binds to receptors on the surface of lymphocytes, which appear after cascade immune buildup. This way, the antigen is saved to memory and the lymphocyte with such a marker becomes sensitized.

After the defense reaction, as waste products of this reaction, strands of RNA are stored as DNA repeats on the genome periphery.

The immune system is resistant to overuse, but it has its limits. At a certain stage, a new program starts. Reparative synthesis with insertion of existing ribonucleotide (encoded by the stored repeats) replaces simple reparation process. Prior to building in, ribonucleotide becomes deoxyribonucleotide with the help of reverse transcriptase. Since the *P53* gene suppresses the activity of the *P53R* gene that codes ribonucleotide reductase, the ribonucleotide has no obstacles to build (as DNA) into one of the growth promoters, where it uses missing promoter for gene amplification [2, 33, 36, 45, 53].

Polynucleotide is created with 20 to 24 ribonucleotides and is an ideal vector of DNA molecular cloning because of its functional targeting and characteristics: being non species-specific, antigen specific, resistant to antibiotics action; its $M_m = 2,86 \cdot 10^6$ Dalton corresponds to M_m of ideal artificial vector, created by genetic engineers [28]. After the very first experiments, genetic engineers were quite aware that by creating recombinants with prescribed properties, they only copy what exists in nature, and they admitted that at their first congress in late 70's. On the basis of their definition of genetic recombination, it is not difficult to draw a conclusion that the creation of a new genotype which is different from the parental one and which transfers acquired properties to descendants, is connected with genetic recombination, such as phenomenon observable in cancerous cells.

Genetic recombination is a process of forming a new genotype, different from the parental one. Recombinant DNA is a DNA molecule, created from two or more DNA sources.

The assumption that the process of cancer cell formation is genetic recombination corresponds to molecular biology definitions [21].

Repair of a break by existing nucleotide is equivalent to vector insertion from additional DNA source which is not associated with genome. The vector is a ribonucleic acid (RNA) originating from cellular immunity. This is precisely why the cancer cell is not rejected by the immune system [31].

In 1970s many immunologists believed that human body most probably will not react to unfamiliar agents of non-organic origin. But the immune system reacts if an agent has active centers for communication with RNA and protein. It only takes 1.5 to 2 years of immune conflict in young people with partially formed and not too *compromised* immune system to lead to AIDS. Different forms and localizations of malignant tumors and different initial terms of their development are observed in children and seniors.

Proposed mechanism of CFS, pre-cancer, and pre-AIDS development

Immune systems react to the action of chemical agents by creating protection. Flu-like symptoms (fever, bone and joint pain, weakness, etc.), which are associated by virologists with pre-AIDS, is an anaphylactic response during an antigen encounter. Duration of non-genotoxic stress defines the duration of latent period. The beginning of AIDS is associated with genotoxic strike and tumor formation. This event defines the inconvertibility of the progression of Th leukemia aplastic form. When a T-helper becomes an immortal cancerous cell, it acquires a characteristic that contradicts its functional purpose in the body – launching an immune response by means of self-destruction.

There are mechanisms in the human body that destroy cells which stop performing their functions. The disease lasts two to five years, during which normal T-helpers are replaced by the cancerous ones, and ends with immune deficiency and the patient's death.

The fact that Pre-AIDS symptoms (review of more than 230 sources) match CFS and pre-cancer symptoms (review of 26 sources and our own observations) makes one think there is a common mechanism in the development of these diseases.

In 2008 in Berlin Dr. Gero Hutter performed bone marrow transplantation in a 28-old patient diagnosed with leukemia and AIDS. The patient recovered. HIV disappeared from all the tissues of his body. No leukemia means no tissue invasion by HIV. This successful treatment supports the leukemia theory of AIDS. (Recent speculations about occasional HIV-suppressive gene transfer with bone marrow transplant do not have grounds).

Discussion

Proponents of the infectious nature of AIDS cannot answer the main question: Why do only Th cells die if the virus is in other blood cells and other tissues of the body? They believe that the markers used for detection of the virus in the genome of several patients is weighty proof of infection, without suspecting that this is evidence of the presence of a malignant tumor, and not an infection. The viral-genetic concept of the genesis of viral tumors (leukemia included), which according to WHO data comprise 15% of all cancers, has been unwittingly confirmed. Its author L. Zilber demonstrated experimentally that the genetic information of a virus is retained in a transformed cell without the complete virus particle being present; the cell continues dividing, retaining its malignant properties.

The tumor hypothesis was not examined for a long time, possibly because of the conviction that leukemia is a disease of immature cells – progenitor cells. B-lymphocyte disease (hairy cell leukemia (HCL)) has been known for several decades, but it was only acknowledged in 2006, when clonal differentiation of activated B-cells made it possible to confirm the degree of their differentiation by immunological, biochemical and cytogenetic characteristics, and not just by morphology. In 2008 a comparative experiment was performed on mice, and it was concluded that mature T-cells are less susceptible to malignant transformation than progenitor cells. This gave rise to the proposal that T-lymphocyte leukemia, like B-lymphocyte leukemia, appeared later than other forms of leukosis due to the greater resistance of mature cells in comparison with immature cells, and not due to the appearance of a mythological virus.

The human immunodeficiency virus (HIV) was "cobbled together" from three viruses belonging to different strains, one lentivirus and one oncovirus. It became necessary to ascribe to them properties of variability and exceptional aggressiveness, which are uncharacteristic for these viruses. The assumptions of the AIDS researchers have not been validated by experiment.

Scientists who participated in the development of viral biological weapons know that a virus's virulence cannot be increased. An experiment by Australian geneticists showed that a non-lethal infection becomes 100% lethal as a result of the discoordination of immunity, and not by increased viral virulence (9).

The assertion that AIDS can be considered an infectious disease only because it undergoes epidemic dissemination is also in error. Many chronic diseases are widespread, but this is no reason to identify them as infectious diseases. Some groups of scientists have attempted to prove that myocardial infarction, hypertension, chronic fatigue syndrome (CFS) and hepatitis C are viral in origin because active immunity is observed in patients. As early as the 1970s, the well-known Russian immunologist Rem Petrov, popularizer of non-infectious immunology, predicted that "virus hunters" who are not familiar with non-infectious immunology would use any activation of cell-mediated immunity as a motive to look for a virus.

The assertion that only a virus can be the cause of a group illness is untenable. Group infections pneumocystis pneumonia (PCP) and lymphadenopathy, which are considered highly characteristic of AIDS, were known long before AIDS appeared. Physicians always considered concurrent non-infectious

factors that lower the body's overall resistance to be the decisive cause. The inoculation of HIV-sensitive monkeys was unsuccessful in producing a complete picture of human AIDS. In addition, only those monkeys that had lived in a research facility for a long period of time fell ill. Their fellow monkeys who remained in the wild stayed healthy. This experiment clearly demonstrated that in addition to a virus other factors exist that determine whether an animal becomes ill or not, and possibly those factors are decisive.

Researchers at the Pasteur Institute who first detected the virus in AIDS patients believed that the virus should be considered the cause of the disease only if it were primary. If a person's immune status changes, an opportunistic virus is secondary. It becomes pathogenic only by reducing immunity.

The immune system of an inhabitant of a large modern city experiences a triple stress. Hypoxic, due to the reduced oxygen content of the air; toxic, as a result of which air pollutants damage cells; and specific, when it is forced to react to constant attacks by these substances by forming antitoxic antibodies and circulating immune complexes (CIC). These causes do not exclude the immune deficit hypothesis associated with overuse of the immune system. It has been assumed that the maturing of progenitor cells does not keep pace with the destruction of Th cells, associated with their physiological role in the body. At the same time, it was felt that no other system in the body has the ability to recover as much as the immune system has. And if after 30 years of observations not one AIDS patient's immune system has recovered under favorable circumstances, and no patient has recuperated, then the hypothesis of overuse of the immune system proposed in 1991 has proven to be false.

The tumor hypothesis remains to be examined. But in order for the leukemia hypothesis to enjoy a more or less sympathetic reception, one more false conviction must be overcome before it is asserted, that is that the disease agent involved in transmission via the blood can only be a virus. In 1966 the American immunologists R. Lerner and F. Dixon transmitted an experimental disease (Sh. Berchanu considered it a model for any immune deposit disease) from a sick animal to a healthy one by transferring serum containing antitoxic antibodies and CIC. Without a virus! It must be concluded that the passive vertical transmission of a disease from a mother to her offspring and the horizontal transmission from one person to another are both possible.

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