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REVIEW ESSAY

AIDS, Cancer and Arthritis: A New Perspective by Phyllis Evelyn Pease, D.Sc., Ph.D. Birmingham, UK: Phyllis Evelyn Pease, 2005. 184 pp. £25.00 (paper). ISBN 0-9550567-0-5, phyllis.pease@orange.fr, http://www.phyllis-evelyn-pease.com/ or contact Pease Associates, Le Bidalou, Audinac, 09200 Montjoie, France. Fax: (33) 561 048109.

This book offers a new way forward in our understanding of AIDS and the mechanisms underlying it, which may be enormously important to future directions for research and treatment. Written by a medical microbiologist with more than 30 years of experience with "hands on" work, especially that involving electron microscopy, it makes extensive use of laboratory-acquired expertise and evidence to question the view that a retrovirus is responsible for the collapse of the immune system seen in AIDS. It describes a possible alternative mechanism in the initiation of AIDS, associated with infective agents but not in a conventional way, and proposes that the same mechanism may also be involved in some forms of cancer and arthritis.

Pease accepts that antibodies currently held to indicate the presence of HIV, as used in the HIV test, can be a genuine signal of immune dysfunction. She presents evidence, however, that these antibodies are in fact autoantibodies, directed against some of the body's own cell constituents, rather than representative of an immune response to an infectious agent. This would indicate that AIDS is essentially an autoimmune disease, a view that found currency in the early years of the epidemic but which fell from favour after HIV was accepted in 1984 as the cause of AIDS.

Pease's research was carried out mostly in the faculty of medicine at Birmingham University, in the UK, where she worked with immunologists and rheumatologists as well as virologists in developing expertise on the life cycles of bacteria, their ability to contaminate tissue cultures, and their possible role in various major forms of disease.

Her first book, for which she was awarded her D.Sc. degree, was called *L-forms, Episomes and Autoimmune Disease*. Published by E & S Livingston in 1965, it is still available in the libraries of leading universities in the UK, the USA, and some other countries. It proposes that episomes—units of hereditary material that can exist as free, autonomously replicating DNA—are sometimes picked up by bacteria and carried in bacterial sub-units, known as L-forms, into the cells of higher animals and humans. This alien DNA can give the infected cell different characteristics, including the ability to produce foreign proteins, sometimes with far-reaching consequences for health.

This was a radical viewpoint at the time of the book's publication, when it was believed that an insurmountable barrier existed between microbial DNA and the

DNA of more highly organised beings. The book was nevertheless widely praised for its "prophetic insight" and the phenomenon was later confirmed by plant biologists, becoming the basis for most of the laboratory techniques of genetic engineering familiar today.

Viable Hypothesis

In 1993, Pease left the UK for Southwest France, continuing her studies at the University of Toulouse. She intended her second book to be a follow-up to the first, adducing evidence for the episome theory of disease from other fields of research, including plant biology and research into cancer-causing organisms. She maintains that the role of bacteria in transporting DNA from one source into the DNA of a related human or animal is still a viable, although so far mostly unexplored, hypothesis for explaining malignancies and autoimmune conditions, possibly including AIDS.

The theory is that when DNA is transferred from one individual to another in this way, and succeeds in colonising cell-control mechanisms, it provides the potential for either malignant or autoimmune disease. In malignancy, normal cell-division and cell-maturation processes are disturbed as the invading DNA produces unwanted growth-regulating proteins. In immunological disorders, the DNA encodes for immune-regulating proteins. These may either be such that the altered cell comes to be seen as foreign to the host's immune system, so that an attack follows on what in reality are the body's own cells, or the altered cell attacks normal cells, mistakenly seeing them as foe rather than friend, as in Graft-Versus-Host Disease (GVHD). Pease proposes that the latter mechanism lies at the heart of AIDS.

She became interested in AIDS and the HIV theory when she found that retroviruses had also been proposed as causative agents of rheumatoid arthritis and other autoimmune diseases. This line of research, she says, has proved entirely unproductive; and her study of the literature on HIV led her to conclude that it has also been unhelpful in understanding AIDS. The search for the cause has been far too narrow, with too little attention paid to the immunological aspects of the disease.

Pease takes the view that a wrong turn was taken in President Nixon's "War on Cancer" in the 1970s, when viruses were made the focus of the huge research effort involved. This not only impeded cancer research, but also gave rise to the HIV=AIDS concept, "which according to a number of investigators is a total misconception. Their doubts are basically concerned with the haste with which certain valid, important observations were adopted by the retroviral cause without due cognisance being taken of alternative explanations and that this state of affairs has been very considerably compounded by the use of uncontrolled techniques." The latter, Pease says, seems to have occurred because biochemists usually have a poor understanding of micro-organisms as living creatures. They tend to regard bacteria as laboratory tools—as bags of enzymes,

or as culture media, for example—and to believe that biochemical techniques are all that are necessary for identifying and isolating viruses. All too often, where AIDS research is concerned, medically trained individuals have adopted the same techniques.

"The result of this simplistic approach is that it has been accompanied by the virtual abandonment of that *sine qua non* for a properly trained microbiologist, the microscope, and in the case of filterable forms of bacteria and viruses this means the electron microscope. Without these aids and the controls that they offer, it has become apparent that what have passed as preparations of pure virions have in fact been contaminated not only with filterable forms of bacteria but also with cellular materials derived from the tissue cultures in which the viruses have been cultured." (Preface, p. vi)

Pease's new book attempts to begin to make good these shortcomings. It is liberally illustrated with microscope and particularly electron-microscope images, including her own preparations. She uses these both in presenting the case that bacteria have a vital role as DNA vectors in human diseases and in demonstrating that "failure to use these tools has led to serious errors of interpretation and the *impasse* that AIDS research in particular has now reached."

The book examines some basics of immunology and parallels between plant and animal cancers in furthering the case that transfers by bacteria of alien DNA lead to human diseases. The text then draws on Pease's lifetime's work in microbiology to drive home her critique of the HIV theory of AIDS and indeed to challenge the whole notion of retroviruses.

Mycoplasma Contamination in AIDS Research

Pease begins by sharing insights into the life-cycles of bacteria, including their ability to change their shape and behaviour in different environments, as living creatures with a considerable range of tactics for survival. Since the 1960s, she says, a tendency to disregard these capabilities has been prevalent, with the result that their potential as contaminants in scientific experiments has been neglected, sometimes with disastrous results.

One such disaster surrounds the role of mycoplasmas in AIDS research. Mycoplasmas are bacteria that lack a cell wall and which can pass through bacterial filters and, hence, "easily escape detection by the untutored research worker." This is particularly likely to happen in research in which tissue cultures are involved. A 1956 report on the phenomenon led to an extensive reappraisal of virus research by the then virological research establishment, including senior investigators in Pease's laboratory in Birmingham. The reason was that various characteristics—especially cell-killing capacity—which had been attributed to viruses were in fact due to the mycoplasma contamination.

The phenomenon was encountered by the group led by Luc Montagnier, HIV's co-discoverer, in 1990, during a search for drugs less toxic than AZT, the first drug used for the treatment of AIDS. Their experiments were on a cell line

in which it was thought that HIV's cell-killing properties had been demonstrated. In fact, when this cell line was treated with tetracycline, an antibiotic, the growth of contaminating mycoplasma was blocked and the cells grew undamaged. "This paper demonstrates simply, elegantly and clearly, a vital piece of information, *i.e.* that *in vitro* HIV is not cytopathogenic," Pease comments.

The customary procedure with contaminating bacteria is to discard the cultures and start again. But in the case of AIDS, that did not happen. Instead, the discovery was interpreted as having unearthed a co-factor needed to explain the variable progression from HIV infection to the onset of AIDS. It was hypothesised that when an individual became infected both with HIV, thought to have come from Africa, and mycoplasmas from elsewhere, that could be the trigger that led to the onset of clinical AIDS.

Pease argues that work led by the USA's Robert Gallo in co-establishing the HIV theory was similarly confounded by a failure to recognise the role of mycoplasma contamination. Gallo claimed in his 1984 papers to have developed a cell line called H9 that allowed HIV to grow. He argued that this was a big advance in that it would allow the putative virus to be studied and a vaccine to be developed quickly—within two years. His explanation for why the cell line kept going despite the virus's purported cell-killing properties was that it had become resistant to HIV.

This is clearly a misinterpretation, Pease says. The true explanation is that the cells became resistant to the contaminating mycoplasma, not to the non-cytopathogenic HIV. This explanation is supported by studies showing that when H9 cells are experimentally infected with mycoplasma as well as HIV, there is no cell-killing effect. Pease also reproduces electron micrographs of an H9 cell culture used as a control for HIV which she says clearly shows mycoplasma elements, growing without any noticeable effects on the cells.

Pease writes: "Unfortunately, this information has been ignored by most AIDS research workers, and ever since the realisation that mycoplasmas have sometimes been closely associated with AIDS, their precise role in the disorder has remained problematical. Indeed, such has been the state of affairs in AIDS research that Montagnier and Blanchard (1993) when discussing the finding that neurotoxic properties initially attributed to HIV could not be reproduced in mycoplasma-free conditions, wrote 'This case clearly shows that the testing of cell cultures and virus stock for mycoplasmal contamination is not a current practice in AIDS research'. This was 10 years after the first recognition of the epidemic, and clearly covers the time during which the most vital early steps of isolation and identification of HIV took place. This flaw in AIDS research has never been addressed seriously by that research community." (p. 24)

The belief that H9 cells are permissive for HIV growth, as opposed to being permissive for mycoplasma growth, has had far-reaching and unfortunate effects in AIDS research, Pease says. It led to widespread use of this cell line in all sorts of research areas, for instance, much of the work on simian AIDS. But with the mycoplasma living freely and inconspicuously in the tissues used to culture

viruses, the scope of confusion will have been enormous. "Indeed it is probable that the use of H9 cells has undermined the findings in untold numbers of research projects."

Pease also presents evidence for mycoplasmas and similar "silent" infections being quite widespread in human blood. Although they do not normally cause disease in people whose immune systems are healthy, their unsuspected presence in AIDS patients is likely to have led to mistakes not only in diagnosing "HIV" disease, but also in interpreting the effects of therapy. Fluctuations in so-called viral counts in response to drugs may be due to alterations in the activity of these micro-organisms. Pease expresses great concern that similar misinterpretations may be putting at risk healthy pregnant women and newborn babies when they are encouraged to take dangerous and even life-threatening anti-retroviral drugs on the basis of studies that have failed to take into account the presence of these bacterial agents.

What Does the AIDS Test Signify?

The concept of HIV as the cause of AIDS was originally based on the belief that the presence in serum of antibodies to two antigens, referred to as p24 and gp120, seen in the blood of people with AIDS and people at risk of AIDS, was elicited by a specific infective agent. The possibility that these antibody patterns were responses to altered self-antigens arising from endogenous processes, most notably graft-versus-host reactions, was not considered, Pease says. This is despite the fact that AIDS is characterised by the production of autoantibodies to an extent comparable to that found in Systemic Lupus Erythematosis (SLE), which has been described as the classic autoimmune disease.

Luc Montagnier and his colleagues at the Institut Pasteur in Paris reported the first presumed retroviral strain, which they thought was associated with the AIDS protein p24, in 1983. A year later, Robert Gallo and his colleagues in the USA published four papers describing similar findings, but with the additional claim that they had found a way to grow HIV in continuous culture, despite its supposed cytopathological effects, such that they had enough antigenic material to develop the first "AIDS test."

Pease accepts that development of tests by the Gallo and Montagnier groups was a great step forward. Their use in screening blood and blood products helps to indicate when risks are present and has greatly lessened the transmission of AIDS via these products. But big questions remain with regard to the nature and origin of those risks and what the tests signify in terms of disease process. Since AIDS is a disease of immune pathology, standard methods used in the investigation of conventional infectious diseases are not applicable. For example, p24 sero-positivity is found in SLE and in chronic bacterial diseases such as leprosy, opening up the possibility that a positive result in the AIDS test is a sign of an immunological aberration "rather than simply a straightforward response to a putative exogenous infective agent."

This problem was made worse by the fact that AIDS investigators have lacked a proper understanding of the materials they have been handling, as well as the fact that they have not realised the extent of the contamination of their supposed virus preparations. In addition to mycoplasma, the materials include tissues derived from cells used to support HIV growth. Bacterial sub-units that resemble virus particles are also present, Pease says. HIV has been assigned properties that belong to the materials from these unrecognised sources. "The whole edifice of AIDS research thus becomes severely undermined."

Even after it became clear that mycoplasmas had contaminated the cultures used by HIV's protagonists, and that they were the cause of the cell-killing for which HIV was being blamed, no one wanted to know. Consequently, these organisms are still causing errors in serology, biochemistry and molecular biology.

Failure to Purify

The core mistake was a reliance on indirect indications of retroviral activity, such as the presence of reverse transcriptase, as evidence for the isolation of HIV, without checking with the electron microscope for what else might be present in supposedly "pure" virus preparations. In 1997, two groups finally took the vital step to correct this mistake, using electron microscopy to examine the material obtained with standard retrovirus isolation techniques—and the results, says Pease, were "an awful indictment of the inadequacy of the methods used in retrovirology." The material was so heavily contaminated with cellular elements as to make it impossible to determine virus characteristics *per se*. Thus, another claim in HIV theory, that "molecular mimicry" between HIV particles and human immune cell antigens might play a role in the disease process, proved unfounded. It is an illusion quite simply due to contamination, Pease says; it is not a question of mimicry, but rather of the particles never having been separated from the cellular elements.

Presumed HIV genetic sequences detected by French and US groups in the mid- to late 1980s were not of pure HIV at all, but rather included a mixture of elements from cells and possibly also from mycoplasma. "This almost certainly accounts for the alleged high rate of mutations of HIV used to explain failures in the search for a vaccine and of the failure to find effective long-term therapeutic agents."

Pease argues that the belated finding from these electron microscope studies that cellular material has never been separated from virus particles casts a shadow, not only on AIDS research, but over retrovirology as a whole.

Since the same methods had been used to supposedly isolate and characterise retroviruses since 1970, when their existence was first proposed, the implication is that at least until 1997 the work was performed by investigators who lacked understanding of the materials they were handling, "and this of course goes hand in hand with potential disasters in interpretations." With the advent of computer graphics, a spurious authority was lent to images of retroviruses by basing them

on well-established structures of other viruses, such as herpes. HIV has been the subject of a great deal of this sort of treatment, Pease says, with images produced as a result of pre-conceived ideas which recent work has shown to be wrong.

The retrovirus concept was originally devised to explain the presence of both RNA and DNA in the life-cycle of some infective organisms implicated in cancer. They were thought to be viruses with a special ability to convert RNA in their particles into DNA, using an enzyme called reverse transcriptase. It was believed that this enabled them to insert their genes into the chromosomes of their host's cells, so that they would become integrated with host DNA.

Research based on this idea lay behind the well-intentioned "War on Cancer." It proved an expensive and disheartening failure. But just as this failure was being acknowledged, AIDS began to be diagnosed in the USA. Designation of HIV as the cause gave the retrovirology field a new lease on life, fueled by billions in funds for research and treatment.

Trying to Save the Theory

Pease's book describes how by the mid-1990s, as facts emerged that did not fit the orthodox HIV=AIDS concept, matters reached a watershed. One school of thought advocated widening the approach to the problem, but a rear-guard action to try to save the HIV theory, enthusiastically endorsed by the leading science journal *Nature* and immortalised in a statement by New York researcher David Ho as "It's the virus, stupid!," led to the introduction of "potentially very dangerous therapies, based on demonstrably unsound premises."

Two papers published simultaneously in *Nature* in 1995, one by Ho's group at the Aaron Diamond Institute in New York and the other by a team from the University of Alabama, claimed that difficulty in finding particles of HIV in the blood arose not because it wasn't there but because it was so lethal: it killed off the cells it infected—CD4 helper cells, vital elements in the immunological defence army, whose decline is characteristic of AIDS—almost immediately. These cells were replaced so quickly by fresh cells that the virus's presence was made almost imperceptible by standard methods. But eventually this war of attrition wore out the renewal process, with AIDS as the result.

The groups claimed to present evidence that by treating patients with potent anti-viral drugs, the virus could be defeated and the immune cells saved. There were high hopes of the possibility of a cure, but these were never fulfilled and today the theory is widely held to have been a misconception. Treatments based on it, despite early claims of miraculous success that were possibly due to an influence on other micro-organisms, have been found in the long-term to cause unpleasant and sometimes fatal side effects. Pease comments: "Although ultimately the individual physicians and informed patients must make the decisions regarding treatment, this is not easy for either, since the drug therapy as a whole is still very much in the experimental stages and the news is not good." She adds that although no conflicts of interest were declared in the

Nature papers "it is interesting to note that several of the co-authors were based at pharmaceutical companies."

The misconception that only HIV could be the infective agent causing AIDS has also bedevilled understanding of the tragedy that affected haemophiliacs and blood transfusion recipients during the 1980s and beyond, Pease indicates. Preparation of the Factor 8 clotting agent at that time involved the pooling of donated blood, and whilst screening was initially of high quality in Britain, in the USA the factor was produced commercially, involving very large numbers of "donors." Some were members of AIDS risk groups, including state prisoners and drug addicts. The extent of this problem was not grasped in time to prevent the export of unsafe Factor 8 to Britain, France and other countries. The consequence was a vast, unplanned human experiment consisting of the injection of large numbers of individuals with disease-producing material, the main constituent of which was derived from other human beings. The evidence seemed to point to a virological cause on grounds that the product had been filtered, but mycoplasmas and related cell-wall–less bacteria can also pass through these filters. This was not considered at the time.

In fact, the latest electron microscope work shows that particles previously presumed to be HIV closely resemble filterable, cell-wall–deficient bacteria, both in shape and in variation of size, Pease says.

Support for Alternative View

The book's final chapter explores the possibility that those particles might be involved in AIDS causation, in accordance with an updated presentation of the episome theory of disease that Pease set out more than 40 years ago.

An observation that supports this theory is that patients with diseases connected with autoimmune disorders, such as ankylosing spondylitis (AS) and Crohn's Disease, have certain tissue types—"self" antigens—that are reactive to a variety of bacteria. Lymphocytes carrying these antigens can become "primed to die" in a way that may be comparable to lymphocyte loss in AIDS. Furthermore, investigators have produced evidence for the existence of a plasmid (a type of episome) in the aetiology of AS which they believe to be connected with this priming process.

With numerous autoantibodies being detected in AIDS patients, considerable resemblance between AIDS and GVHD was noted even before HIV was discovered. Since then, studies in mice and monkeys, in particular, have strongly suggested that AIDS is a GVHD.

For example, monkeys become protected from "simian AIDS" by vaccines consisting of cellular antigens only, irrespective of whether the HIV homologue, SIV, is present. The studies have also demonstrated that the antibodies p24 and gp120, thought to show the presence of HIV, are related to the GVHD process. Pease says it seems to be cell damage caused by immunological conflict with cellular material that causes the appearance of the characteristic AIDS

antibodies. Mice in which the same disease is induced experimentally become positive for these antibodies—with no virus involved. They also develop wasting, gut infection, opportunistic infections, skin rashes, and lymphocyte abnormalities of the kind seen in AIDS.

With the latest generations of "AIDS tests," Pease says, detection of p24 is a good indication that an individual either has AIDS or is at risk of AIDS. However, she emphasises that the antibody can also be due to a self-limiting condition, as opposed to a sign of inevitable progression to advanced AIDS. It is now recognised that many people who test "HIV" positive do not develop AIDS. Variations in the composition of the putative episomes could contribute to variations in the course and severity of the disorders, which can range from rapid and life-threatening to chronic and relatively benign.

When animals are injected with antigens from the same species, various autoantibodies are elicited, but these are normally self-limiting. One human equivalent to this could be a mild graft-versus-host reaction resulting from continuous acts of anal sex, in which antigens from sperm can escape into the bloodstream of the recipient. These antibodies can remain in the bloodstream for months or even years.

It is therefore important to take account of the clinical condition of the patient before diagnosing AIDS, including what is happening to their CD4 cells, the lymphocytes seen to be in decline in AIDS.

Pease says that at least part of this decline is explicable in terms of a conflict between "self" antigens on a person's own lymphocytes and differing ones to which an individual has been exposed through procedures such as transplantation, transfusion of blood products, and use of contaminated hypodermic syringes for vaccination and drug injection, as well as anal sex.

Africa may have suffered especially from iatrogenic AIDS through dangerous medical interventions, including mass vaccination programmes carried out by poorly trained individuals. Recent work by researchers from several universities has provided impressive evidence that the heterosexual route for transmission of AIDS in Africa has been grossly exaggerated and that iatrogenic factors have been very much underestimated. Outbreaks of AIDS in China have been associated with dangerous practices involving the mass pooling and sale of blood for therapeutic purposes, often within villages, where victims are likely to be related and therefore to have similar blood-groupings, thus increasing the risk of GVHD disease.

Political and Financial Pressures

With so many flaws evident in the "HIV" theory, how has it managed to retain such a firm hold on scientific and medical opinion? "Even some leading establishment investigators have admitted that there has been a great deal of bad research on AIDS," Pease writes. Some leading scientists—notably Peter Duesberg, Professor of Molecular Biology at the University of California at

Berkeley—questioned the theory almost from the start, seeing it as the result of "a hunch based on expediency" rather than sound judgement.

However, from the onset of the epidemic, the AIDS problem was influenced by political and financial matters to a degree unprecedented in the field of medical research, she says. Those aspects had a considerable influence on the course the research took and in eliminating even well-informed dissent.

"In fact, most mainstream investigators now recognise that AIDS is not a simple virus disease comparable with the great bacterial and viral epidemics of the past, but even so, research continues almost exclusively as if this were the case. There are two reasons for this—one is that most of the funding in the search for therapies and vaccines for AIDS is not only conceptually but also commercially based, which means effectively that AIDS research and HIV research are synonymous. There is thus a considerable amount of pressure on research workers to direct and interpret their findings in a way that encourages the renewal of their grants. As far as what must now be described as the third generation of investigators is concerned, this presents no difficulty because the commercialisation of science is now a fact of life, and in any case, this generation has been taught that the cause of AIDS has been settled as a self-evident truth—that things have moved on—new technology has confirmed the original work—and that anyone who does not accept it is mad, bad or both. The second—scientifically more telling—factor is that since AIDS appears to be an infectious disease there does not seem at present to be a more persuasive explanation other than that an infective agent is involved, and currently, HIV is the only candidate. The result of this intense concentration on HIV is that some areas of AIDS research have become very esoteric, and for many investigators, including myself, some of the research work, especially in the realms of molecular biology, has become almost impossible to follow, let alone evaluate, and seems to be very far removed from the curing and prevention of the disease. The volume of resulting publications is now so vast that it is excessively difficult to grasp anything resembling the whole of the increasingly narrowly specialised picture. On the other hand it is all too easy to see that the promise of effective therapy that came in the mid-nineties has not been fulfilled, and the guarantee of an effective vaccine, notoriously made in 1984 by the then US Government Health Chief, Margaret Heckler, has now disappeared into the unforeseeable future." (p. 112)

Education Needs Widening

Pease concludes that the most pressing need now in the AIDS epidemic is education. This should not be confined to the promotion of condom use, though unprotected, promiscuous sex could clearly pose a risk. For the undeveloped world, it especially needs to focus on the bad medical practices that account for many deaths. And in the developed world, it needs to be understood and recognised that the evidence does not support the HIV concept, even though this

has been marketed as established truth so effectively that there is now a world-wide demand for "anti-retroviral" drugs and for a prophylactic vaccine against a virus whose existence, in the form currently perceived, is not supported by the evidence.

"Paradoxically, the fact is that AIDS, which began as a small allegedly controllable epidemic, has now become a pandemic for which no truly long-term effective therapy has been found and no effective vaccine has emerged. This has led, not to a reconsideration of the role of HIV in AIDS, but to an increase in funding and investment for the search for almost certainly unattainable goals."

All the evidence tells us that AIDS is not in the category of disease that can be controlled by the "magic bullet" approach to fighting infections, Pease asserts. However, the secondary effects of immune insufficiency—rendering the system unable to counter common agents that are easily dealt with by a healthy immune system—can be combated with classical antibiotics. "It is these antibiotics that need to be promoted, in the present state of our knowledge, rather than treatment with dangerous, expensive so-called anti-retrovirals of doubtful value, which may give the impression of efficacy through acting in a non-specific, inefficient way against secondary infections."

Pease's book is the most authoritative and microbiologically precise account to date of the failings of the HIV theory of AIDS. Her alternative, episome theory, does not at present have the same level of scientific evidence as her HIV critique. This is not surprising, since the retrovirus theory has been so influential in securing funds and attention and has been censorious of other points of view. However, the case that the central mechanism of damage in AIDS is an autoimmune process is strong. In view of the new lines of inquiry and therapeutic potential the theory offers in AIDS as well as other major illnesses, it should be urgently investigated.

Pease concludes that "such lines as I have discussed, however bizarre they may seem at first sight, should be followed up. If I am right about the role of bacteria as vectors of alien DNA (this applies to the various disorders discussed in this book as well as AIDS) then a new approach to vaccines might prove effective as well as being cheap to produce and innocuous. It is above all important that the preconceptions that have hindered AIDS research be put aside. As long as it is necessary to warn research workers that they must not mention any doubts they may harbour about the connection of AIDS and HIV, the nature and relevance of retroviruses or the role of iatrogenesis, or allow the young to hear different views for fear of ridicule and rejection, and even loss of livelihood—we are not likely to advance further in lines of research where these phenomena must be looked in the face and proved true or false on the evidence." (p. 171)

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