

Racism and AIDS: African Origin Theories of HIV-1

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The AIDS epidemic, first discovered in the United States in 1981, has caused a great deal of speculation with regard to the origins of both the HIV-1 retrovirus and the early pathways for the epidemic itself. The African origins theory is the most widely accepted origin theory for HIV-1 in the West. This theory is based upon six assertions, all of which either lack evidence or, when evidence has been present, these assertions have been contradicted. The African origins theory is unsubstantiated. The African origins theory is based not upon scientific logic but rather upon victim-blaming, the attempt to define "the other" as the cause of disease, and racism.

From where did Acquired Immune Deficiency Syndrome (AIDS) and the HIV-1 virus come? What are the ultimate origins of this retrovirus and the illness it causes? What are the hidden arguments behind the search for the origins of both HIV-1 and the AIDS epidemic? We will examine these questions in the following essay.

AIDS was first discovered in the Spring of 1981, although it was encountered clinically by medical practitioners much earlier than this date. From the earliest encounters with the AIDS syndrome, and with what would later be known as the HIV-1 retrovirus, assumptions were made as to the origins of the disease and the pathways which the illness had taken and was taking. Thus, early in the epidemic the syndrome of illnesses associated with the immune suppression caused by the HIV-1 retrovirus was known as GRID, Gay Related Immune Deficiency. This designation, although certainly not the first for the syndrome, helped give it a lasting association with gays in the North American mind.¹

It was a simple step from this name to the lifestyle interpretations and the epidemiological investigations which were to follow. These were to include the infamous "popper" (amyl nitrate) investigations and association,² which was to divert attention from the viral cause of AIDS

for some time and to provide a wall behind which the blood products industry would hide in order to continue their large profits without interruption by the danger of a blood borne and transmittable cause for the AIDS illness.³ The result was literally thousands of hemophiliacs infected with the HIV-1 virus from use of viral infected blood products (Factor VIII). Assumptions concerning origins and pathways, assumptions concerning who is at risk for HIV-1 infection, when placed into policy and practice in a social context, can be costly not only in terms of dollars but also in terms of lives.

Thus, from the earliest days in this epidemic, there has been a continuous and often growing debate concerning the origins of the retrovirus and the consequent risk to individual groups from its infection. This continuing debate may be divided into two questions which are often conflated and confused during the discussion.

The first question is truly a question of origins and a question of history. From where did the HIV-1 virus, the viral causative agent of the AIDS syndrome, originate? Where did this retrovirus come from? Since the AIDS syndrome appears to be of recent emergence, when and where did the causative agent evolve? It seems, especially since this is an epidemic which arose in the 1970s or 1980s, that this question should be capable of being answered. It should not be necessary to look into the incompletely recorded past, as it is with the sexually transmitted disease of syphilis, for instance, to understand the origins of AIDS.⁴ Was it, as in the classic Small Pox viral origins, an animal crossover? If this is the case, how long did the retrovirus exist in the animal populations and from what animal did it cross over? If it is not an animal crossover, did HIV-1 evolve in the human population? Syphilis, for instance, is often thought to have been a developmental result of a previous human disease which was not sexually transmitted (Yaws). It should be recognized that, while these are scientific questions, they contain explosive emotional, social, and political overtones. These questions cannot be asked without a certain tension occurring and without larger implications being included in any answer.

There is a second and related series of questions. These second questions are often associated and confused with the first; they concern both the present and the past spread of HIV-1 infection. They ask the following: What are the pathways of the AIDS virus, the HIV-1 retrovirus? What human populations have been differentially struck by this virus and the consequent AIDS illness? What are the characteristics of these populations which have made them particularly susceptible to this differential impact? What has made AIDS appear the way it has epidemiologically? These are not questions of ultimate origins but of the intermediate pathways of the epidemic. They may be viewed as closely related to the first question of ultimate origins but are one step removed from such a first, and historical, question.

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It must be understood that these questions also have great emotion attached to them, and nowhere is this more clearly demonstrated than in the AIDS epidemic. Original groups who suffer from the infection, particularly if the epidemic is a sexually transmitted disease, can be stigmatized and victim-blamed. Again, the long standing debate concerning the origins, European or North American, of the syphilis bacillus are an example of this. The emotional content of epidemic origins can, and is, assigned to early infected groups, such as has been so clearly the case with gay males in the United States. Thus, a debate concerning the early pathways of a sexually transmitted epidemic is not purely a scientific question, just as the ultimate origins of the retrovirus itself is not a purely scientific question.

Why ask these questions at all if there is such a stigma potential? In fact, if one can discover the immediate precursor virus to an infectious and deadly virus, there are possibilities that this earlier ancestor may not be virulent or deadly to humans. It may be possible to use the ancestor as a basis for a vaccine and the foundation for prevention of the subsequently evolved virus. This is the scientific justification for the search for origins. It is, ultimately, founded upon the historic model of Jenner's vaccine, derived from Cow Pox, the ancestral virus to Small Pox which was not virulent to humans. Cow Pox was used to provide the basis for a vaccine for Small Pox. We have seen Small Pox eradicated due to the widespread campaign to use this vaccine in the farther corners of the world. If this model were applicable to the HIV-1 virus, it certainly would justify the search for origins.

This, then, is the scientific justification for the search concerning the origins of the HIV-1 virus. Certainly, the search for the earliest human population which became infected with HIV-1, and developed AIDS as a consequence, is also capable of such a scientific justification. Finding this early history of the epidemic would also open the possibility of discovering an early precursor and developing a vaccine.

During the course of the following essay, we will examine the African origin theory of HIV-1. Thus, we will be looking at the answers that have been given to the initial question mentioned above, the question of the ultimate origins of the HIV-1 virus. In this essay, we need to look at the scientific justifications for these theories but, as I will argue more extensively in the latter sections, we need to look at the hidden content of these theories. What is it that they are saying between the lines of their arguments and theories? Is logic and science the sole basis for their approach, or, rather, are there hidden assumptions and hidden political agendas contained within these scientific pursuits? Needless to say, I will argue at length that there are, as Sylvia Tesh has termed them in her brilliant book, hidden arguments behind all these investigations.⁵ These hidden arguments are very significant and important, at least as significant as the arguments which are visible.

Ultimate Origins Theories for the HIV-1 Virus

Even before the pathogenic causative agent of AIDS was isolated and identified, with a test developed for its presence in 1985, theories for the origins of the as yet unidentified HIV-1 retrovirus were advanced. Perhaps none have been as widespread or as enduring as the theory that the HIV-1 virus originated in central Africa. One cannot work in the field of AIDS research and have one's work known by the public without being questioned frequently concerning the African origins theory of the AIDS virus. It is one of the earliest topics brought up in any conversation concerning AIDS and the AIDS epidemic. When students in a large introductory sociology course were tested as to their knowledge of AIDS at the University of Utah, for instance, seventy-nine percent responded that the HIV-1 virus originated on the African continent when given a choice of all continents and a last choice of "unknown". This is the theory most widespread and most accepted among educated, and I suspect, uneducated, North Americans. It appears early in the epidemic in both the popular press and in scientific and popular scientific works by "experts".

This theory took some time to develop, partly because it was not until 1983 that the AIDS syndrome was clearly identified as existing in Africa. This occurred as a result of a number of wealthy Africans traveling to Europe for medical treatment for intractable illnesses for which they had sought treatment, unsuccessfully, in Africa. The intractable illnesses were, in fact, opportunistic diseases, characteristic of AIDS, and the syndrome was soon recognized in these patients from the African continent. Theoretical speculation did not take long concerning the origins of AIDS. In April 1983, in a letter to the famous British medical journal, *The Lancet*, Jane Teas of the Harvard School of Public Health advanced the theory that the African swine flu virus might be the precursor to the AIDS virus. This was, of course, before the retrovirus causing AIDS had even been identified and a test developed for its presence! Teas also made the Haitian connection for the origins of the AIDS epidemic at the same time:

Closely paralleling the onset of the first cases of AIDS in 1978 in Haiti was the first confirmed appearance of the African swine fever virus (ASFV) also in Haiti, in 1979...In 1976, ASFV was confirmed in Cuba and all pigs were killed. The island remained disease-free until 1980, when the virus reappeared, coincident with the arrival of Haitian refugees...When an infected pig was killed and eaten either as (uncooked or uncooked) meat. One of the people eating the meat who was both

immunocompromised and homosexual would be the pivotal point, allowing for the disease to spread amongst the vacationing 'gay' tourists in Haiti.⁶

The Haitian connection was suggested initially in the epidemic by the discovery of thirty-four cases of AIDS among Haitian immigrants to the United States in 1981, practically coincident with the discovery of AIDS in the United States.⁷

Needless to say, complaints flooded into the journal as a result of this ingenious letter and its creative theory. It was widely speculative, totally without any epidemiological support, and, in later investigation by researchers from Belgium and Holland, proved completely unfounded. The Haitian connection so made in a mere unresearched letter, in association with the above mentioned thirty-four cases among Haitian immigrants to the United States, was sufficient for the Centers for Disease Control, and the public, to designate Haitians as a high risk group, however. In fact, in retrospect, we know that the African swine virus is not a retrovirus at all, and therefore unrelated to the AIDS virus which is, of course, a retrovirus. Because they are fundamentally of differing families of viruses, African swine virus could not have been the precursor for HIV-1 but the die had been cast. In subsequent research on Haitians with AIDS, it was further determined that all of these Haitians were involved with the tourist trade with North America. None of the individuals who were early cases of Haitian AIDS had ever even met an African. It appeared that AIDS came to Haiti from North America, rather than the reverse.⁸ However, Africa and Haiti had been selected as the focus of speculation concerning the origins of the AIDS epidemic and the viral causative agent. The future would hold many such unsubstantiated theories concerning the African origins of the virus and the epidemic.

Early investigations by researchers in Africa in 1985, using the recently developed enzyme linked immunosorbent assay test (ELISA test), which detects the body's immune system reaction to the HIV-1 virus, seemed to show very high rates of HIV-1 viral infection in many areas of central, eastern, and southern Africa.⁹ Particularly, high prevalence rates were detected in several African cities and, subsequently, HIV-1 infection was detected in rural areas also. Such high rates of infection, it was argued, must prove that the disease had been present in Africa for some time in order to spread so extensively and reach such high prevalence rates. Certainly, it was asserted, to have been so widespread in 1985, HIV-1 and AIDS must have been in Africa for at least a decade or more. Also, testing of early blood samples and stored blood samples from the late 1950s seemed to indicate a very early date for the presence of HIV-1 on the continent of Africa, at least as early as 1959. A pregnant woman was found to be HIV-1 positive in Kinshasa, Zaire, in 1970. A study of the sera saved from several

"remote" tribes in Zimbabwe, Liberia, and Kenya in the late 1960s and early 1970s "confirmed the presence of HIV-1 in two specimens from the Mano tribe of Liberia".¹⁰ It is often asserted that these are the earliest evidence of AIDS and HIV-1, certainly earlier than in North America. As Shannon, Pyle, and Bashshur assert:

With the notable exception of KS and other mysterious symptoms found in the frozen tissue of a 16-year-old black American male who died in 1969, no serum samples stored in the United States prior to the 1970s have been found to be seropositive. On the basis of serological studies there is some indication that HIV infection may have emerged earlier in Africa than in the United States. However, the rapidly rising incidence of cases in Africa also suggests a new epidemic of infection perhaps as recent as 40 to 50 years ago.¹¹

If these arguments are true, then the next question to ask is: Where and how exactly did HIV-1 originate on the African continent?

There are many creative and very imaginative responses which have been made to this question. Let me try to outline the basic African origins theory as developed in the popular and scientific press from 1983 onward.

The African origins theory usually starts with monkeys, particularly, green monkeys in equatorial Africa. These green monkeys are infected with a simian immuno-virus known, appropriately enough, as simian immunodeficiency virus (SIV). This retrovirus does not cause any immune deficiencies in this African equatorial green monkey, but when injected into other species of monkeys it causes an AIDS-like illness. It is this retrovirus which is most often cited as the precursor for HIV-1 in humans. How did the crossover from monkey to human occur? This, as one might expect, is where real theoretical creativity occurs.

It is generally speculated that the crossover from monkey to humans occurred due to some sort of cultural/sexual practice in the equatorial regions of Africa. In its most lurid presentation, this crossover occurs because of voodoo or shamanistic sexual practices, such as the injection of monkey blood from the green monkey to enhance sexual performance, pleasure, and excitement by some groups of Africans.¹² In other versions in the popular media and in oral mythology in North America, the green monkey is consumed uncooked by Africans and this provides a crossover route very similar to the route posited earlier for ASFV by Teas. Another variant for the crossover event posits a monkey bite as the crossover pathway.¹³ Certainly there is room for many creative theories here, especially since it is clear that such a crossover event is impossible to document or prove; it thereby becomes a

subject of speculation and mythology rather than an actual event. At any rate, whatever the crossover methodology, the green monkey retrovirus is seen as the ultimate precursor to the HIV-1 virus and AIDS. According to this theory, the retrovirus, famous for its fluidity in terms of its chemical/genetic structure,¹⁴ must have mutated during one of the contacts in Africa with humans and became the HIV-1 infectious agent for the AIDS epidemic.

The story has variants from here, just as there are variants in the crossover path. In some versions, the retrovirus, having once crossed over and mutated, lay dormant for various periods of time before breaking out into the general population in Africa due to social disruption such as warfare, massive migration, or agricultural/ecological disruption. Once it broke out from its endemic rural enclave, however, its movement into the general African population is evident, according to the prevalence studies on the continent conducted from 1983 onward. From the African continent it spread to the world, eventually striking North America.

This certainly sounds like a brilliant thesis, and it is asserted that it fits many of the facts and much of the historic evidence from the development of the AIDS epidemic. The basic outline presented above has been advanced by numerous popular journals as well as some "authorities" in the field of scientific research and virology, such as Robert Gallo.¹⁵ Let us look closer, however, at the basic assertions of this theory. The fundamental assertions are:

1. HIV-1 and/or AIDS appeared first on the African continent, probably in the 1950s.
2. The evidence for #1 above is present in stored African blood samples and HIV-1 prevalence studies from rural and urban Africa.
3. There is evidence that HIV-1 infection and AIDS have been present for some time on the African continent, perhaps in a rural area remote from contact with the rest of Africa.
4. The precursor retrovirus, the green monkey SIV, is closely related to the HIV-1 virus. Genetic research demonstrates this close relationship and demonstrates the mutation or descent of the SIV green monkey virus into HIV-1.
5. There must be some crossover method or event which allowed this SIV retrovirus to make the leap from animal, presumably the green monkey, to human beings, where it became HIV-1.
6. The high prevalence rates of HIV-1 infection and AIDS provide evidence for the earlier spread of HIV-1 infection and AIDS in

the African context.

Not all of these six assertions must be true for the African origins theory to stand, but some of them must be verified. For instance, certainly the earliest appearance of HIV-1 on the continent of Africa must be true if it originated there (#1, #2, #3, and #5). If the crossover theory from the green monkey (#5) is correct, then the genetic relationship between HIV-1 and the green monkey SIV must be true (#4). If this hypothesis is not true, then there must be some early relative of the HIV-1 virus on the continent. Perhaps some other SIV?

Are these six assertions true and verified by the scientific literature? No. In fact, not a single one of these assertions has clear support or evidence in the scientific literature. Most of the six assertions have been directly contradicted by research in the past five years. Let us take the above statements one by one and examine them, beginning with the assertion that the earliest cases of AIDS were in Africa.

Frequently, when unusual cases occur, unexplained in causation and unusual in clinical course, they are documented, reported, and written up. This material is then sent to various medical journals, there to remain for posterity's reference and possible use. In a very important article, appearing in the *Review of Infectious Diseases* in 1987, David Huminer, Joseph Rosenfeld, and Silvio D. Pitlik retrospectively examined these anomalous cases in medical literature published from 1950 through 1978, looking for reports of illnesses which would fit the clinical definition of AIDS.¹⁶ It needs to be noted that, even without sophisticated laboratory confirmation, the clinical criteria correctly delineate AIDS in most cases. In fact, in Africa, where laboratory confirmation is both too expensive and too difficult to obtain, the clinical definition of AIDS is quite valid and reliable. What did these three researchers find in their retrospective study of the medical literature? They found nineteen cases of illness which fit the clinical definition of AIDS over the course of this twenty-eight year period. The first such case occurs in 1952 and was reported in 1953.¹⁷ From where was the first case reported? From the United States. As Huminer et al. state:

Eight of 19 cases were reported from the United States. Two reports each came from Canada, the United Kingdom, and West Germany. Isolated cases were reported from Sweden, Denmark, Belgium, Uganda, and Israel.¹⁸

Almost half of these cases occur in the United States and the rest occur in various other western countries, with the exception of Israel and Uganda. The case in Israel first appears in 1969. The first appearance of an AIDS case in Africa occurs in Uganda in 1973. This is more than two decades after the first appearance of AIDS in the United States.

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This study is the best available on early cases of AIDS, and it is hardly proof of the earliest appearance of AIDS on the African continent. In fact, it proves nothing of the sort. It should be noted that this study also refutes such origins theories for HIV-1 as the famous CIA theory which posits a CIA creation of the HIV-1 virus. The creation of this virus by anyone is out of the question since techniques for gene section splicing and DNA recombinant work were developed decades later than 1952, which is the earliest appearance of AIDS.

There are a number of complexities in the cases reported by Huminer, et al., however. First, all of the laboratory data are not present, and this is especially true of immunosystem data for these patients. In most cases there are no tissue samples saved and the presence of HIV-1 cannot be verified irrefutably. Tissue samples are present from a mysterious case in St. Louis in 1968. This case, resulting in the death of a young Black man in that city, was definitively determined to be AIDS because of the presence of HIV-1. This still predates the first appearance of an African case by four years, according to Huminer, et al. It should be asserted again at this point, however, that a clinical case definition of AIDS, such as used in this study, has been found to be highly valid and reliable when diagnosing AIDS in the absence of laboratory facilities.

A number of those who suffered from these early cases had traveled extensively. It is interesting to note that the article specifically mentions travel to Africa, although many of these individuals clearly traveled to a number of other areas of the world. Any conclusions concerning the geographic origins of the AIDS from which they suffered is wildly speculative and unsupported.

Another complication, which may be introduced into this data, is the level of surveillance in the medical delivery system in the various areas of the world. In other words, if the United States system is much more extensive and complete, the AIDS syndrome may simply trip the surveillance wire earlier on the North American continent. This may account for the earlier cases in the United States. The difficulty with this assumption is that numerous physicians, operating in the African context, have stated that AIDS-like illnesses were not present in any detectable numbers previous to about 1979.¹⁹ Often not reported in the scientific or popular media are the results of HIV-1 blood tests of African blood which have been done and have *not* supported the African origins thesis. Work done in the mid-1980s, which used three different screening tests for the presence of anti-bodies to HIV-1 in blood from male and female children and adults from Uganda, found no evidence of HIV-1 infection in any of the blood. This triple level test is much more reliable than the single tests often used in other reports and strongly supports the absence, in any significant numbers, of HIV-1 in Uganda before 1975.²⁰

It should be noted that Uganda, of all African countries, had, during the 50s, 60s and 70s, a quite sophisticated system of medical reporting. This country is one of the few countries in the world, and certainly in the Third World, to produce a complete disease atlas of its territory in the 1970s.²¹ Uganda, of all countries in the Third World, is the most sophisticated in medical surveillance terms. The western assumption of primitiveness and lack of surveillance is not justified.

The fact still remains that, in terms of the medical literature, there is no proof of the early existence of AIDS in Africa. Existing evidence, in fact, points to the earlier existence of AIDS, and possibly HIV-1, on other continents, in other areas, and in the West and the United States. What about the early blood tests which confirmed numerous cases of HIV-1 infection on the African continent?

The early blood tests mentioned in item #2 above were contaminated with large numbers of systematic false positives. The ELISA test does not function reliably in the African context because the test is confounded by both stored blood (in any context) and blood which contains malaria antibodies.²² The early blood tests which showed high rates of HIV-1 infection in cities and residual rates in rural areas where essentially meaningless since they were performed on both stored blood and blood endemically infected with malaria. Particularly the residual levels, often referred to as endemic and mentioned in #3 above, in the rural areas, are well within the false positive range of the ELISA test. In other words, the endemic rural areas of HIV-1 infection in Africa have no validly documented infection levels at all. The evidence for #2 and #3 above is, from a scientific standpoint, universally recognized as unreliable and, in fact, nonexistent.

The idea that somehow this endemic infection, present in sheltered and isolated enclaves of rural Africa, broke out into the larger community on the continent during the 1960s or 1970s, is also ridiculous on its face. There are really no such isolated areas on the continent and have not been for many decades. The world is a much more integrated and interacting place than this model would have us believe, and includes, of course, Africa and its rural communities.

The assertion of the genetic relationship of HIV-1 retrovirus to the African equatorial green monkey simian immunovirus (SIV) is without doubt one of the major assertions of the African origins thesis (#4 above). The mutation of SIV into HIV-1 has been the theoretical animal crossover for the African origins theory for almost a decade. Of all facts in the AIDS oral mythology of North America, none is so widely asserted as the relationship of the green monkey virus to HIV-1. Are HIV-1 and the green monkey SIV closely related? Is the green monkey SIV the precursor virus to HIV-1, thus proving the origins of HIV-1 itself? The answer is clearly and emphatically no.²³

It is, and has been, clear for almost five years that HIV-1 and SIVs are not closely related. They have some similarity; they are both retroviruses. HIV-1 and SIVs share some genetic similarity, it is true. But neither one is the descendant of the other, and that has been clear for some time.²⁴ Further, HIV-2, which is present in western Africa and causes a modified form of AIDS which is less virulent, has only about seventy-five percent genetic similarity to SIVs of the green monkey. HIV-1 also has been compared to SIVs, particularly in the green monkey, and it shares only about forty percent similarity with the closest SIV. It appears that HIV-2 is not directly related by descent to HIV-1 either, and the relationship of either HIV-1 or HIV-2 to SIVs is complex and not readily apparent.²⁵ One should note that chimpanzees and humans share a great deal more genetic similarity than this; in fact, chimpanzees and humans share well over ninety percent genetic similarity, and yet they are neither descendants, one of the other, although it is theorized that they do descend from a common ancestor.

Credible researchers are willing to admit that we do not know enough about retroviruses in Africa to make any valid statements concerning their relationships, one to another, nor to make statements concerning their relationships to HIV or its descent.²⁶ Some research indicates that the origins of SIV among green monkeys in Africa may be very ancient and these retroviruses may have co-evolved with the monkeys as long ago as 10,000 years.²⁷ These conclusions, if true, make the origins of HIV-1 much more complex and distant than at first supposed.

The geography of this infection is incorrect for an SIV crossover, also. If HIV-2 is the most closely related to SIVs, then why is the HIV-2 virus concentrated in western Africa rather than eastern Africa? Eastern African is where both the green monkey is resident and where HIV-1 has its highest prevalences. The geography of these distributions is wrong for an animal crossover from the green monkey to humans for HIV-1. As one text admits: "The situation may be much more complex and speculative than currently described."²⁸ This complexity is sometimes introduced, however, in the scientific literature in order to attempt to save the SIV to HIV-1 descent and the African origins thesis.

In order to attempt to save the HIV-1 descent from some monkey virus, the animal crossover theory and the African origins theory for HIV-1, the scientific literature has begun speculation concerning a mutual exchange amongst a number of SIV retrovirus, all infecting a host at the same moment and mixing their genetic materials in a sort of wild orgy of DNA splicing. It is posited that a number of SIVs may have infected, benignly it is admitted, a human host all at the same time, then exchanged their genetic materials in some complex way, thus creating the resulting HIV-1 in that particular host. This multiple combined and

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mutated virus then is transmitted and is the HIV-1 virus. Or is it the HIV-2 virus? The speculation on this matter, it is admitted in the scientific literature, is completely unsupported by any foundation of research or evidence. Given the complexity of the interchange, it is unlikely that this particular event will ever be documented let alone scientifically proved or established. The scientific literature has moved into the realm of mythology, the mythological great exchange or the massive DNA splicing orgy, if you please, as the event which must have occurred. This mythological creation, this theoretical crossover event, exists in order to save the African origins thesis and the green monkey crossover. This theory of HIV-1 origins is pure and unadulterated speculation—flight of fancy. It is interesting that recent popular articles have asserted that science has now determined that HIV-2 and, ultimately, HIV-1, are definitively related to SIVs in Africa.²⁹

Almost all reports and research on the ancestry of the HIV-1 and HIV-2 virus concentrate on the African connection. There are numerous retroviruses in the world today, not the least of which are feline retroviruses, equine retroviruses, and bovine retroviruses, all of which occur in the West with considerable frequency. All of these occur in animals which are in close association with humans in the West. None of these retroviruses have been investigated to near the extent that SIVs in Africa have been investigated. It seems to be a case of looking determinedly for the answer in one place, sure that it must be found there and not elsewhere. Perhaps Richard and Rosalind Chirimuuta have stated the underlying factors most effectively:

Because scientists found it so difficult to imagine that white people could infect Africans with AIDS and not the reverse, such a possibility has never been seriously investigated.³⁰

In conclusion, the evidence has very definitely *not* been found for the animal crossover theory of the origins of HIV-1, despite the popularity of this theory in the Western press and in the AIDS mythology of North America. Thus, the hypothesis in #4 above is completely unproven.

The assertion in #5, that some crossover event has allowed HIV-1 to move into the human population, has been the source of some of the most shameful anthropological research to be conducted in this century. One can find many assertions, particularly again in the popular press, that monkey blood is used for many purposes on the African continent. It is interesting to note that none of these practices have been well documented in the scholarly literature by reputable researchers. The "crossover" event has been the focal point for virulent Western racism with regard to African cultural practices for over a decade. Needless to say, there is no well-researched or documented example

of this crossover event, nor could there be since there is no evidence for a precursor retrovirus on the African continent in the first place. Thus, the hypothesis in #5 above is wild speculation and totally unsubstantiated.

Finally, we may examine the sixth assertion above. This assertion advances the present high prevalence rates of HIV-1 and AIDS on the African continent as evidence for the longstanding existence of the virus in the African context. It must be admitted that, when allowances were made and interpretative practices were adjusted to the African context for HIV-1 testing, there remains at present a relatively high prevalence rate of HIV-1 infection in Africa. This high infection rate is concentrated in the "AIDS Belt" in central, southern, and eastern Africa, with some of the highest infection rates for HIV-1 in Uganda and surrounding areas. Certainly Zaire also has alarmingly high infection rates with HIV-1. It has been argued that these infection rates, much higher than anywhere in the West in "pattern 1" AIDS, are evidence of the longer existence of AIDS and HIV-1 on the African context. Does the higher prevalence rates in Africa constitute proof of a longer existence for HIV-1 on the African continent? No.

There are many reasons why the HIV-1 virus may have spread into the African population more extensively than in the population in Western countries. Not the least of these reasons may be a migrant labor system which provides not only a means of contracting sexually transmitted diseases at high prevalences in African populations, but also provides a ready and extremely efficient amplification/transmission system for sexually transmitted diseases (STDs).³¹ It has never been asserted that syphilis or gonorrhea originated on the African continent. Yet very high rates of syphilis and gonorrhea, in fact a rampant epidemic of these diseases, occurred in the very same areas of Africa during the 1960s and 1970s. The social factors, such as a migrant labor system, which helped create these extremely high rates of STDs in previous decades, are still operative in the AIDS epidemic. It has further been asserted that long standing cultural practices with regard to sexuality³² as well as military selection during the recent Civil War and unrest in eastern Africa and Uganda³³ have both, in the past decade, accelerated the spread of HIV-1 in the African context. If any or all of these means of amplifying the transmission and spread of STDs are operating, and I would assert that some, at least are operating, then the high prevalence rates of HIV-1 in eastern, central, and southern Africa are proof of a social structure spreading STDs, not of a biological origin of the retroviral cause of AIDS. Is the sixth assertion above proven? No.

In a recent *Scientific American* article by the two epidemiologists, Roy M. Anderson and Robert M. May, it is interesting to see how the African origins of the AIDS epidemic is supported.³⁴ They assert that the high levels of infection with HIV-1 in Africa are proof of the African origins of the epidemic and, in fact, assert that crossover thesis for the

virus itself. As Anderson and May state

The AIDS virus almost certainly evolved in Africa...In the worst-afflicted urban centers in Africa, 20 to 30 percent of pregnant women are infected with HIV. This level of infection has sometimes been attributed to socioeconomic conditions. It is more likely that this high rate of infection is a consequence of the length of time over which the virus has been spreading in these areas: the epidemic is simply further advanced.³⁵

Therefore, where the African epidemic is concerned, the high levels of infection have a biological, not a social origin. The reason for these high levels, according to May and Anderson, is that the disease is biologically further advanced in the epidemic cycle. How then do we explain its high levels in some areas and among some populations in the developed world? As Anderson and May state:

The epidemic developed quickly in the early 1980s among intravenous drug users and male homosexuals in the U.S. and Western Europe. This rapid advance undoubtedly resulted from the introduction of the virus into communities having behavior patterns ideally suited to viral spread: drug injection and frequent intercourse with many different sexual partners.³⁶

What is happening here? The simple answer is that, in terms of their treatment of Africa and the United States, when faced with high rates of infection, Anderson and May shift their means of explanation depending upon which continent they are addressing. When it comes to Africa, there is a biological explanation which supports the African origins thesis; when they are explaining the high infection rate phenomena in the United States and Western Europe, they move to a social/behavioral explanation. Why not a social or behavioral explanation for the high rates of infection and AIDS in Africa? It would undermine their presumption of an African origins for the HIV-1 virus.

Anderson and May never mention that the earliest cases of AIDS are found in the West over two decades before they are seen in Africa. They must be aware of this, however, because they go to some length to try to explain why there is no evidence of early cases of AIDS in Africa. How do they explain this?

The human virus could have been slowly spreading in parts of Africa for 100 to 200 years, possibly even longer. It could be that in these much earlier times, the complex beginnings of the epidemic (as localized flickerings

in rural areas) could not be detected against a high background noise of infection and disease. Or it may be that ancestral forms of HIV in humans did not lead to AIDS.³⁷

The authors of this article try to advance the trip wire argument combined with an argument concerning ancestral forms. Do we have any evidence of this ancestral form or its development? No. Might the same argument be made for the presence of such an infectious but non-disease causing HIV in North America? Yes, but Anderson and May do not make the same arguments for North America that they make for Africa, despite the evidence of earlier cases in North America. They are desperately trying to support an African origins thesis, no matter what, and if this requires shifting arguments and the refusal to apply the same standards of discussion to both continents, well then, so be it.

What remains, then, of the African origins theory of the HIV-1 virus? Little, if anything. After almost a decade of research, all we have are a few totally unsupported assertions, a number of unscientifically founded speculations, and considerable contrary proof. The essential hypotheses upon which any African origins theory must be based are simply unproven and in some cases, directly contradicted, by the evidence. The theory is still advanced in popular media and in scientific and popular/scientific forums, however.

In June of 1988, a letter appeared in the *Lancet*, concerning an early case (beginning about 1966) of AIDS in a Norwegian sailor and his family.³⁸ The authors were all physicians from various respected departments in Norway. The letter cited the Huminer, et al. article concerning AIDS in the pre-AIDS era and discussed this early Norwegian case of AIDS which killed the Norwegian sailor and much of his family. The letter specifically mentions that this sailor traveled to Africa, although it also admits that he traveled to a number of other areas in the world and contracted STDs at least twice, presumably from any one of these areas. The article makes a major point of asserting "proof" of the first case of AIDS in Europe, ignoring cases in the Huminer, et al. article it cites that precede this "first" European case by almost a decade.

In fact, in a 1989 postscript to their book denouncing the African origins theory (which was first published in 1987), Richard and Rosalind Chirimuuta state:

In the two years since the book's first publication, it has been gratifying to find that much of the evidence for an African origin for the Human Immunodeficiency Virus (HIV) has not stood the test of time. Most importantly, the African green monkey is generally now no longer thought to harbor a precursor to HIV that

crossed the species barrier, and other animal viruses, particularly retroviruses in sheep and cattle, are under consideration...

Although the scientific evidence for an African origin has been found wanting, the scientists are only reluctantly abandoning their favorite hypothesis and are considering the alternatives with little enthusiasm.³⁹

Unfortunately, the Chirimuutas are too optimistic. The popular and scientific literature has not abandoned the African origins thesis, and had only abandoned it temporarily by 1989 in the face of mounting contradictory evidence. The question becomes, then, why is this the theory which is promulgated in the popular Western media? Why is this mythology the theory which is prominently accepted by "educated" Westerners? The basis for the acceptance of the African origins theory of HIV-1 in the West **must** be based not on a scientific rationality but on other reasons. What are those reasons?

Conclusions

The African origins theory of HIV-1 is based upon a number of hidden justifications, all of which are not "rational" in the scientific sense. The first justification is the tendency to victim-blame for such a serious sexually transmitted disease. It is a commonly observed tendency in many societies to blame the victim of a disease for the disease itself. This tendency is nowhere more prevalent than in sexually transmitted diseases, where it can be seen most clearly in the practice of separating "innocent" victims from "guilty" victims.⁴⁰

There is a further justification. That justification, or hidden argument to use Tesh's terminology, is based upon the desire of individuals in the west to see the HIV-1 infection and AIDS as affecting others, even to the point of originating with others. It has commonly been observed that most diseases are named for a region other than the one assigning the name. Thus, flu viruses are usually named by North American researchers for Asian locations. STDs are often called by the French diseases of the Germans, and by the Germans, diseases of the French, and so on. This is also the case with AIDS. As Susan Sontag has observed

One feature of the usual script for plague: the disease invariably comes from somewhere else...But what may seem like a joke about the inevitability of chauvinism reveals a more important truth: that there is a link between imagining disease and imagining foreignness. It lies perhaps in the very concept of wrong, which is archaically identical with the non-us, the alien. A polluting

person is always wrong, as Mary Douglas has observed. The inverse is also true: a person judged to be wrong is regarded as, at least potentially, a source of pollution.⁴¹

This tendency, long observed in many contexts, is also exacerbated by the combination of racism and fear of the Third World. In the first instance, Africa can be portrayed as the "dark" continent. A continent of unhealthy practices and people, of sexual license, and disease. All of the stereotypical responses of Westerners to racially "colored" people can be used, subconsciously and consciously, to support these hidden assumptions. Again, Sontag has stated it well

Thus, illustrating the classic script for plague, AIDS is thought to have started in the "dark continent," then spread to Haiti, then to the United States and to Europe, then...It is understood as a tropical disease: another infestation from the so-called Third World, which is, after all where most people in the world live, as well as a scourge of the tristes tropiques. Africans who detect racist stereotypes in much of the speculation about the geographical origin liminal connection made to notions about a primitive past and the many hypotheses that have been fielded about possible transmission from animals (a disease of green monkeys? African swine fever?) cannot help but activate a familiar set of stereotypes about animality, sexual license, and blacks.⁴²

In this conception, we can explain the longevity of the African origins myth not by its scientific basis, since this basis is and has been for some time largely non-existent, but by the hidden reasons or arguments for this origin of the HIV-1 virus. The African origins of the virus fulfills the social need, in a highly conservative era, to victim-blame in STDs as well as to see others as the source of the illness. Further, the other is conceptualized not only as another race but also as the Third World in an era when United States military might is "downsized", made highly mobile, and turned from its former task of combating the Soviet Union to defeating dangerous uprisings and insurgencies in this very area called the Third World. The Fall of 1992 was the 500th anniversary of the Columbian establishment of the division between First and Third worlds, and it is this split, this division, which is expressed in the persistent existence, despite lack of scientific support, of the African origins theory for the HIV-1 virus.

In the face of the myth creating potential surrounding the AIDS epidemic, social and natural scientists can only ask that hidden assumptions be made apparent. Those interested can ask for research, if not

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with fewer biases, at least with explicit ones. Perhaps it is time to investigate, for instance, the possibility of an origin for HIV-1 and the AIDS epidemic in some other location than Africa and the Third World. Perhaps it is time to investigate other possibilities for an animal cross-over, including those possibilities in the First World as well as the Third. In fact, it is time to investigate the distinct possibility that this disease began in the First World. Hopefully, this will not be accompanied by the same sort of victim-blaming ideology that we have seen so apparent in the investigation of Third World origins for the AIDS epidemic and HIV-1.

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