AIDS, THE SCIENTIFIC MIND-SET AND
THE ‘TECHNICALISATION’ OF TWENTIETH CENTURY SCIENCE

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Abstract
This paper provisionally explores some the limitations of the HIV/AIDS model in terms of the transformations in the nature of scientific inquiry that have occurred at least since the end of the Second World War. A central question to be addressed is how to differentiate between the nature of scientific thought as a way of systematising our knowledge of the world and the political and economic system in which it is now embedded, which dictates certain research priorities and probably has also changed the quality of the practitioners of science. In this regard, it takes as a major point of departure the writing of the great geneticist, Erwin Chargaff, who spoke eloquently of the pre-war years --long before the issue of AIDS ever arose-- when "the technicalization of biology had not yet won
its shallow victory." Chargaff's concerns (about the mind-set of scientists) will be addressed as a background to the AIDS issue and as a step toward a different approach to AIDS which can give greater attention to the kinds of social, economic and political questions that scientific enterprise, as it is today, may be unable to address.

INTRODUCTION

Scientific reasoning provides a way of correcting the ideological assumptions on which the HIV/AIDS model is based; but not if those assumptions go unchallenged. Scientists have to see the value of the challenge. So, what stands in their way? I would suggest that one of the main things, in the case of AIDS, is the way that thinking about it has generally been separated into what appears to be two mutually exclusive domains: the one epidemiological; the other, sociological. As a result, the science of AIDS is built on incomplete data.

But, this is not particularly surprising. In the decades after the eminent 19th century German pathologist, Rudolf Virchow, wrote that “Medicine is social science or it is nothing,” medical science generally took a markedly different road,
becoming increasingly detached from concerns about the social conditions in which the victims of disease lived. Virchow’s radical perspective was over-whelmed by the professionalisation of science in general and of medicine in particular, which reflected the advance of Western capitalism and the accompanying transformation of science, medicine and research into sources of profit.

This is not to say that this process was un productive. Between the late 19th century and the 1920’s, the labs of inventors such as Thomas Edison and Alexander Graham Bell were transformed into the research and development branches of great multinational corporations and there is no doubt that, backed by enormous capital resources, they could yield impressive results. Between 1937 and 1998, for example, eleven researchers won Nobel prizes for work done while they were at Bell Labs, which was sold by AT&T to Lucent Technologies in 19__ (Lucent Technologies 2002b). But, as Arno Penzias, the co-discoverer of the back-ground radiation of the “big bang,” noted of the years after receiving the Nobel and becoming executive director of the Communications Sciences Research Division and, then, Vice President of Research of Bell Labs (a position he occupied for the next fourteen years): "While we [will] continue to pursue scientific excellence in fundamental
areas, most of our scientists seek breakthroughs in targeted areas." That is to say that the best scientists are not only increasingly likely to work for giant corporations—to have access to the resources that enable research at the frontiers of knowledge—but, to pursue their investigations, however imaginatively, within what is ultimately a commercial setting.

Few scientists have commented as openly and poignantly on the transformation of science as mind-set and practice that this entailed as Erwin Chargaff. Like Penzias, Chargaff’s family were refugees from Naziism; and, like him, he also embodied a pre-war European sensibility—which was also apparent in such scientists as Einstein, Hans Bethe, Isidor Rabi, Victor Weisskopf, and Wolfgang Pauli. They were all born in Europe, most were of Jewish background and their science had a strong philosophical dimension. And all of them—except Chargaff—came together in that watershed moment of modern science, the Manhattan Project. In regard to which, several important points need to be underscored. First, that they all purposefully participated in one of the most momentous scientific enterprises of the 20th century, one which set a pattern for modern science as a collective project focused on a specific set of technical objectives defined by non-scientists, policy-makers or business interests. Secondly, that they all
tended to view the project as a supreme, even enjoyable technical challenge—a view that was summed up Freeman Dyson, when he wrote: “they did not just build the bomb. They enjoyed building it. They had the best time of their lives while building it.” (The Manhattan Project Heritage Preservation Association, Inc. 2002) But, thirdly, and perhaps most importantly, most of them reacted to what they had created with moral despair, which was famously captured in J. Robert Oppenheimer’s quote from a verse from the Bhagavad Gita: "Now I am become Death, the destroyer of worlds.” With the passing of their generation, however, Western scientists would continue to enjoy the technical challenges, but without the guilt.

One can, of course, see in retrospect that the origins of a technicalised science—and the demise of conscience—long pre-dated the Manhattan Project. Associated with the new demands made upon science in the competitive industrial capitalist era at the end of the 19th century, it was embodied in the life and work of one of the earliest Nobel laureates, the German chemist Fritz Haber. When he began his career, Germany, without colonies, needed to find artificial means of securing resources that Britain, France and The Netherlands secured from their colonial fiefdoms. Among the most important of these in the early 20th century was nitrogen fertilizer and Haber would win
the Nobel Prize for Chemistry in 1918 for inventing the process –later elaborated by Carl Bosch (who received the Nobel Prize in 1931)-- for developing the technology to produce ammonia under high pressure. Subsequently, Haber became the Director of the Institute of Physical Chemistry in Berlin.

After Haber was appointed the Chief of the chemistry section in the War Department for Raw Materials, he became interested in the use of chlorine gas and developed a way of delivering it on the battlefield which circumvented the Hague Conventions of 1899 and 1907, which Germany had signed and ratified, and which had limited the use of such weapons. Even Haber’s son Ludwig would later write of how “unscrupulous” his father became in these years when he became, in effect, the originator of modern chemical warfare. Indeed, after the war, he continued to develop Germany’s poison gas potential. But, in 1919, after Allied inspectors of his Institute halted research on chemical warfare, Haber switched to research on agricultural pesticides chemical warfare against agricultural pests. He established a firm, the German Society for Pest Control, which developed a product that would later be used during the Second World War by the SS at Auschwitz and other death camps. It was called Zyklon B
It is likely that Haber, an intense German nationalist, saw these developments as more than a purely technical challenge. But, the point is that science per se provided no moral or intellectual immunity for him—or many others—against engagement in such technical projects. Science was already embedded in the political economy of the nations of which such scientists were citizens and they accepted—sometimes hesitatingly, sometimes (after the fact) with remorse, and, increasingly, with no apparent emotion except the joy of meeting the challenge, with too little regard for the human costs or the social consequences.

Chargaff, who found a professional haven in the illustrious chemistry department of Columbia University, was among the few who openly spoke of this insidious transformation in the nature of scientific work and in the ways of thinking of scientists that had characterised the 20th century. One of the true giants in his field, whose work on nucleic acids made possible the Nobel prize that was won by Watson and Crick, Chargaff would write, in 1980:

I have watched, with increasing dismay and even hopelessness, the ever more rapidly changing scenery of scientific research and also the change in the type of actors who are overcrowding the stage.
He went on:

[T]he financial powers and the means of production, that is, the laboratories, etc., have become concentrated in ever fewer hands...the individual scientist can no longer have a voice. In other words, science has become thoroughly politicized, a playball of power networks in which such expressions as "the search for truth" or "the benefit of humanity" must sound unconvincing, and even ridiculous, since everybody knows what and who are behind them. The distance between an Einstein or an Avery and a Vice-President in Charge of Research is very far indeed. They may use the same words, but they mean different things. In my opinion, there is no denying that the face of science has changed more in the last 15 years than it did during all its previous history.

If science in general has increasingly focused on technical solutions, it is perhaps not surprising that medical science has tended to distance itself from Virchow’s vision. Epidemiologists and medical researchers generally fail to regard disease in terms of its social and economic dimension. In so doing it has also constructed a model of the efficacy of its technical intervention -and especially its contribution to mortality decline in the West-- which, as Thomas McKeown, the historian of population health and professor of social medicine at the University of Birmingham, pointed out in The Role of Medicine: Dream, Mirage or Nemesis? (1979), contrary to both popular and professional wisdom, largely pre-dated medical interventions and was, in fact, largely attributable to improvements in the economic environment and, especially, to
better nutrition. Based on this, his last book, *The Origins of Human Disease*, proposed a tripartite categorisation of disease into 1) prenatal diseases; 2) diseases of poverty; and 3) diseases of affluence (MacKenbach 2004:225).

McKeown’s thesis fits well with medical historian Charles Rosenberg’s view that “A disease is no absolute physical entity but a complex intellectual construct, an amalgam of biological state and social definition.” (quoted in Packard and Epstein 1991:782), a view that should direct our attention to the way that the latter may create such a forceful, yet subtle context that few scientists actually question their initial assumptions --or those of granting agencies== or the economic, social or political interests which have animated them.

**PELLAGRA**

By the end of the 19th century, the creative tension between the new germ theory and the older models of disease –the miasma, etc.–was more or less resolved. Against the background of an increasingly technical approach to medicine, almost everyone was looking for a unique pathogen that caused a specific disease. In this context, Koch’s famous postulates were an important pre-cautionary tool –then and now-- to prevent correlation from being confused with cause (cf.
Duesberg 19__)]. Yet, even so, the concept of a single pathogenic agent had powerful adherents among conservative forces hostile to a more dynamic, processual perspective on disease.

The best example was pellagra, a disease which was spreading rapidly through the Southern United States in the decade before the First World War and was widely regarded as caused by an infectious agent, albeit one which was often linked by eugenicists to a hereditable disposition to infection on the part of the poor (Chase 1977:201-205). This interpretation was definitively challenged by the research of U.S. Public Service physician Joseph Goldberger, who demonstrated that pellagra was a dietary deficiency caused chiefly by poverty (Chase 1977:205-207). Yet, Goldberger’s conclusions were resisted; and as Chase notes, “Between 1914, the year Goldberger discovered the cause of pellagra, and 1928, the year of his untimely death…the reported pellagra deaths multiplied eightfold.” (Chase 1977:212) But, the situation was actually far worse, because pellagra cases were severely under-reported and, in many instances, the condition predisposed people to die from a wide range of other diseases (Chase 1977:212). In particular, in many cases, throughout the U.S. South before the Second World
War, pellagra, hookworm and malaria all interacted to produce the observed patterns of morbidity and mortality.

In the case of hookworm, one should be quite emphatic about the role of material deprivation. Infection in humans by helminth nematode parasites through contact with contaminated soil—usually because people lack shoes—is due to intestinal blood loss. But, in fact, “hookworm disease” occurs when urin deficiency anemia results from blood loss exceeding dietary intake of protein and iron (Hotez et al. 2004:800).

Burkitt’s Lymphoma

Today, of course, we should have no problem placing pellagra or hookworm in McKeown’s category, “diseases of poverty.” But, consider a rather less obvious and more complex disease—Burkitt’s Lymphoma—which was first described in 1968 by Denis Burkitt at Makerere University, and the epidemiology of which still remains obscure (Chase 1982: 327). It has long been known that BL, which is most commonly found in East African countries such as Uganda, has a decided relationship to a human herpes virus known as Epstein Barr. But, at the same time, “The correlation between the geographic and climatic distribution of holoendemic malaria and endemic BL in equatorial Africa has long been recognized and malaria is
regarded as an important co-factor in the disease.”

(Cardy, Sharp and Little 2001:300) But, the situation is even more complex:

Studies in endemic areas indicate that BL is closely associated with EBV. However, this association does not explain the geographical distribution of BL since the majority of tumours in low-risk areas do not appear to be associated with EBV. Nor does it explain its relationship with age or the predominance in males. Given the ubiquitous nature of EBV and the geographical variation of its association with BL, other factors are clearly involved. These may include dietary habits, malnutrition, exposure to certain plant species, arboviruses, other infections and genetic susceptibility. The factor most discussed is persistent and heavy infection with malaria, since the geographical distributions of holoendemic malaria and BL are similar. Changes in the transmission of malaria have been associated with changes in the incidence of BL, although the relationship is unclear and sometimes contradictory. The association may depend on the transmission intensity of malaria, rather than the simple occurrence of falciparum malaria. Again, additional factors must be involved since large numbers of people in equatorial Africa are infected with both EBV and malaria, yet few develop BL. The possible role of arboviruses has received relatively little attention. Clustering of BL is compatible with many etiologic models including involvement of an infectious agent, a carcinogen, genetics, selective population movement or simultaneous operation of multiple factors (Cardy, Sharp and Little 2001:303).

Many considerations suggest that Burkitt’s Lymphoma may be regarded, in McKeown’s terms, partly as a pre-natal disease, affected by certain genetic factors, and in part as a disease of poverty, with a possible relationship both to malaria, the
distribution of which is closely associated with the colonial and post-colonial development history of Sub-Saharan Africa, and to poverty and poor nutrition (Cardy, Sharp and Little 2001:3030).

With time, the socio-economic context of a wide variety of diseases –most recently, for example, cervical cancer in Mexico (Palacio-Mejia 2003)– has been found to offer new insights into their distribution and prevalence.

The question to pose, then, is why, in the light of what we know of diseases such as pellagra or hookworm and Burkitt’s Lymphoma, why in the light of recent research that suggests a strong poverty factor in the pattern of diseases such as cervical cancer, modern medical science reverted to a narrow technical search for the “cause” of AIDS. If the reason is similar to which kept Goldberger’s insights into pellagra from gaining headway in the early part of the 20th century, it raises fundamental questions about whether we can expect the scientific mind to help free us from the constraints of the dominant economic and political ideologies of our time, until it itself is freed from orthodox assumptions.
In the case of Burkitt’s Lymphoma, an additional point needs to be made. Cardy et al. (2001:303) note that there has been little recent research on BL in areas of high-risk, while “most recent publications are molecular studies, such as those of chromosomal breakpoint location and gene expression, and deal with BL in a Western setting.” In other words, scientific research has tended to emphasis a highly technical aspect of a potentially wider, more complex problem, at the expense of other, potentially more productive lines of research where genetic, microbial and social factors interact. Has this happened again in the case of AIDS?

**AIDS in Sub-Saharan Africa: Beyond HIV**

Underdevelopment in much of Sub-Saharan Africa has long been ascribed to a population "problem.” (Ross in press; cf. Ross 1998) A 1991 report by the UN Population Fund (UNFPA), for example, noted that, while Kenya's economy since independence had been among the best performers in Sub-Saharan Africa, "the benefits of a strongly growing GNP have been markedly reduced by population growth." (UNFPA 1991:95). The report also attributed declining agricultural production to the pressure of population on the environment which, it argued, drove people into the cities where unemployment is rife (Ibid: 96). Little attention was paid, however, to the fact that the seeming
evidence of Malthusian pressures—such as migration out of rural communities—was, in fact, closely tied to the way that colonial economies confined indigenous Africans to inadequately resourced areas, while their post-colonial successors did remarkably little to redress this legacy.

For a number of reasons that I have discussed at length elsewhere (Ross in press). Malthusian views—rooted in colonial assumptions about African sexuality—carried over into the independence era. In Kenya, for example, the government demographer, John Blacker, located in the Ministry of Planning and Development (MPD), argued that the slowing of Kenya's population growth rate was essential to ensure the new country's economic development (Watkins and Hodgson 1998:17). According to Ajayi and Kekovole, such concerns "have been a recurring theme in national development plans from the first (1966-70) to the current 8th development plan (1997-2001) (Ajayi and Kekovole: 115). Most importantly, they were given the imprimatur of the New York-based Population Council by its famous mission to Kenya, at the request of the MPD, in 1968 (Bondestam 1980:159-160). The Population Council's report unambiguously asserted that any effort to narrow the gap between people's aspirations and economic development depended
on a program directed toward a decrease in the rate of population growth." (quoted in Bondestam 1980:162).

Nevertheless, until the early seventies, the Kenyan economy was regarded as fairly successful, with an annual GDP growth rate of over 5 percent, even though the benefits of economic growth were not equitably distributed and the incidence of poverty was high. But, according to the Eastern Africa Multidisciplinary Advisory Team (EAMAT) of the International Labor Organization, the situation worsened considerably during the following decades. A recent study shows that the level of absolute poverty in 1994 was around 44 per cent; for the urban areas it was 29 per cent, while for the rural areas it was nearly 47 per cent (1999:9).

As a result, according to a recent paper by the Central Organisation of Trade Unions (COTU) in Kenya, the country entered the 21st century facing what is arguably the worst economic crisis since the country gained independence.

In spite of this, by 1998, the International Monetary Fund was taking credit for an "African economic renaissance," claiming, along with the World Bank, that the structural adjustment policies it had applied to countries such as Kenya had paid off. This claim is widely disputed, but the idea that African
countries are economically on the upswing defines the prevailing framework within which the Bank and OECD countries typically address a wide variety of policy issues facing the Third World. Among these is AIDS. Because the IMF and the Bank are now curiously optimistic about the economies of countries such as Kenya, they are disinclined to view AIDS in Sub-Saharan Africa as a function of poverty. On the contrary, where the World Bank was saying in 1983 that rapid population growth was "the single most important obstacle to sustaining rising living standards in Kenya over the long term," today, when population growth is lower, the Bank now maintains that the main impediment to sustained economic prosperity in Sub-Saharan Africa is AIDS. This position is exemplified in a 1999 draft report by the Human Development Sector of the Africa Region of the World Bank, *Why Strategies to Reduce Poverty in Africa Should Focus on HIV/AIDS and Sexual and Reproductive Health*. Because it attributes AIDS exclusively to HIV and HIV transmission to sexual behavior, the paper effectively reduces underdevelopment in Africa to a reproductive health issue, which hinges on individual responsibility.

In terms of the epidemiological history of the region, the orthodox Western prognosis for AIDS in Africa represents nothing very new. Fourteen years ago, Packard and Epstein
(1991) noted that it echoed expectations about the consequences of syphilis (another disease that was characterised as sexually transmitted, although African syphilis often turned out to take non-venereal, such as yaws) voiced earlier in the 20th century when, for example, “a leading British expert on syphilis…concluded in 1906 that ‘As things are at present, the entire population is in danger of being exterminated by syphilis in a very few years, or of being left a degenerate race fit for nothing.’” (quoted in Packard and Epstein 1991:772)

It was in this intellectual environment, a colonial legacy, that, as Epstein and Packard note, “Early discussions of AIDS in Africa developed.” (p. 773) Thus, from early on, when it was necessary to consider why the spread of AIDS was characterised by a relatively balanced sex ratio in Sub-Saharan Africa, compared to the overwhelming number of cases among homosexual men in the West, this contrast was largely explained in terms of certain assumptions about African sexual behavior. It was a theme that became “the central focus of medical inquiries into the problem of AIDS.” (p. 774) and it was certainly where the research funding was. One waited a perilously long time for medical science to question the legitimacy of these assumptions –until David Gisselquist and
his colleagues, in an article published in 2002 in the International Journal of STD and AIDS, demonstrated that there is little correspondence between patterns of HIV prevalence (not to mention AIDS) in Sub-Saharan Africa and those of sexual behaviour.

This strongly suggests that we must seriously reconsider the epidemiology of AIDS, especially in the case of Sub-Saharan Africa. At the heart of such a rethink should be a focus on the economic, social and environmental consequences of debt and structural adjustment and their cumulative impact on the human immune system and its capacity to respond to the resurgence of many of the diseases that are currently subsumed under the rubric of AIDS. Why? The first cases of AIDS were documented in Africa in 1982. It was first reported in Kenya in 1983. In the rush to blame AIDS and its predicted impact on the same sexual practices which had long been regarded as the source of the region's "population problem" and diseases such as syphilis, there were only a few writers -- among them Meredith Turshen of Rutgers University -- who braved questioned (cf. Turshen 1988) whether we should not also be looking at political economic factors and to the vulnerability of people whose immune systems were compromised by poverty and malnutrition. A similar position has been taken by the World
Development Movement in a 1999 report, *Deadly Conditions? Examining the Relationship Between Debt Relief Policies and HIV/AIDS*, which notes that, "While not restricted solely to poor people, AIDS is a disease of poverty, marginalisation and social and economic injustice." This is certainly likely to be the case in a country such as Kenya where, according to the World Bank in 1995, "half of the population was...unable to consume a minimum requirement of food and essential non-food commodities."

Turshen, however, still subscribed to the conventional view that the sexual transmission of HIV caused AIDS. But, University of California retrovirus specialist, Peter Duesberg, has made a powerful argument over the past fifteen years that this is very unlikely to be the case. While not disavowing the idea of HIV causation, David Gisselquist and his colleagues, in an article published last year in the *International Journal of STD and AIDS*, nonetheless lend significant support to Duesberg's position by noting that there is little correspondence between patterns of HIV prevalence (not to mention AIDS) in Sub-Saharan Africa and those of sexual behaviour. This strongly suggests that we must seriously reconsider the epidemiology of AIDS, especially in the case of Sub-Saharan Africa. At the heart of such a rethink should be a
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The World Development Movement noted in 1999 that, as of three
years ago,

One third of children in Africa are significantly
malnourished. UNICEF suggest that SAPs have
contributed to the widespread deterioration in the
nutritional status of children, pregnant women and
lactating mothers in rural and urban areas
undergoing structural adjustment programmes.

The implications for the spread of AIDS diseases are clear,
for, as The Merck Manual notes,

Malnutrition can also seriously impair the immune
system. The malnutrition may involve a deficiency
of all nutrients, or it may involve primarily
proteins and certain vitamins and minerals...When
malnutrition results in a weight that is less than
80 percent of the ideal weight, the immune system is
usually somewhat impaired. When the weight is
reduced to less than 70 percent of ideal weight, the
immune system is usually severely impaired (Merck
1995-2000)

Thus, in 1998, in its State of the World's Children Report,
UNICEF stated "that malnutrition impairs the immune system of
at least 100 million young children and several million pregnant women --none of them infected by HIV."

Among other causes of immuno-suppression are diseases such as malaria and tuberculosis, both of which are on the rise globally, in developed countries such as England (Elender, Bentham and Langford 1998) as well as in developing countries such as those of Sub-Saharan Africa. As a result, the WHO already had estimated, in 1990, that one-third of the world’s population was infected with tuberculosis, which caused one-quarter of avoidable deaths among young adults (Parry and Davies 1996) Trends in tuberculosis prevalence seem to be closely related to material deprivation (including poor and over-crowded housing, poor diet) and, although the WHO has designated tuberculosis as an AIDS disease, recent evidence from England certainly suggests “that AIDS has little influence on the level of tuberculosis mortality in the wider population.” (Elender, Bentham and Langford 1998:673). In the countries of Sub-Saharan Africa, the rise in tuberculosis is likely to be related to adverse economic development policies since the early eighties, including the impact of structural adjustment. Yet, the conventional HIV/AIUDS model clearly has prevented research in that direction.
In such areas as Sub-Saharan Africa, an increase in malaria—which is on the rise in many parts of the world—can be attributed to the spread of anopheline mosquitoes (the malaria vector) into new niches that are being created by the effects of economic development strategies such as deforestation.

Deforestation, in turn, is rarely the result of solely local actions or even of national policies, but owes much, as Solon Barraclough (1992) has concluded, to the "rigid insistence on certain kinds of monetary, fiscal, trade and privatization policies by the international financial institutions in the name of stabilization and structural adjustment" (which have led to an increase in commercial agriculture, logging, loss of fuel subsidies or simply to the financial inability of the state to manage and protect forest resources. In general, as Susan George has concluded, the unrelieved burden of Third World debt has forced poor countries with significant forest reserves to generate export earnings by cashing in on their natural resources.

In some places, World Bank-promoted expansion of non-traditional export crops has also meant a "continued assault of pesticides," which has led to widespread mosquito resistance, at the same time that malarial parasites have also become
resistant to conventional drugs. This is a special problem for developing countries whose shrinking public health budgets cannot assume the costs of newer, more effective treatments. Meanwhile, many of the agro-chemicals used in commercial agriculture in such countries themselves pose a serious threat to the integrity of the human immune system.

There is no doubt that there have been sporadic but important efforts to highlight the impact of structural adjustment on the rise of ill-health in Sub-Saharan Africa. Yet, in general, even this argument, while understanding that malnutrition itself weakens the immune system, assumes that this simply makes people "more vulnerable to viruses such as HIV," when the deterioration of the immune system is precisely what AIDS is.

The failure to recognise that the cumulative impact of the structural determinants of malnutrition and adverse environmental change on the immune function of the global poor may be enough to account for the rise of AIDS—and of immunological dysfunction generally—in developing countries such as Kenya—is also a failure of medical science to set its inquiries in a broad social context, rather than a narrow technical one.
In general, historians of science and medicine have tended to confirm the insights of Virchow and McKeown. But, Western, science by adhering to a far more restrictive medical model, has not only failed to challenge the conventional AIDS model; it has effectively allowed non-scientists to continue to set the AIDS agenda. It has allowed the World Bank to remain committed to the conventional view that AIDS is the result of patterns of sexual behavior and to suggest that the key to the future economic well-being of Sub-Saharan Africa is a change in the sexual practices of its people. This view is echoed in a recent WHO report by Harvard’s neo-liberal guru, Jeffrey Sachs, and his colleagues (Sachs et al. 2001), which ascribes the high prevalence of "HIV/AIDS" in sub-Saharan Africa to particular sexual patterns and practices and which regards the costs of AIDS as one of the chief impediments to African development.

CONCLUSIONS

The question today is whether science is too much embedded in the system which generates the dominant ideology and whether its priorities are too subservient to that ideology to allow the critical nature of scientific reasoning to help us to question conventional thinking and its human costs and consequences. There is some suggestion that headway is slowly being made toward a more productive interpretation of AIDS.
Though a virologist and not a social scientist, Duesberg has pointed the way, along the lines suggested by McKeown’s earlier work, by emphasising that the disparate epidemiological patterns in the U.S. and Sub-Saharan Africa are unlikely to be explained in terms of a single HIV-caused AIDS pandemic. As he wrote in 1992:

The hypothesis that human immunodeficiency virus (HIV) is a new, sexually transmitted virus that causes AIDS has been entirely unproductive in terms of public health benefits. Moreover, it fails to predict the epidemiology of AIDS, the annual AIDS risk and the very heterogeneous AIDS diseases of infected persons. The correct hypothesis must explain why: (1) AIDS includes 25 previously known diseases and two clinically and epidemiologically very different epidemics, one in America and Europe, the other in Africa (Duesberg 1992:201).

Thirteen years later, nothing has changed—except that there is both more evidence in Duesberg’s favor and even more hostility toward his dissident views—views which embody precisely that aspect of the scientific mind which Chargaff lamented the passing of. No wonder that Basu, Mate and Farmer were prompted to write, in a letter to Nature:

Given the strong correlations between poverty and AIDS, one might question the priorities of those scientists who have chosen to battle with dissident colleagues rather than making the main thrust of their campaign the epidemiology of a disease worsened by sustained indebtedness (2000)

in countries in Sub-Saharan Africa especially.
Since Duesberg’s first interventions in the AIDS field in the late 1980’s, the sexual transmission of HIV has been called into serious question from different quarters, at the same time that it has become increasingly clear that the distribution of AIDS diseases depends on a number of factors, among which malnutrition is preeminent. Since malnutrition is a source of immuno-suppression in its own right, the putative role of HIV is necessarily becoming less essential. The time has surely come then for us firmly to situate AIDS within the broader context that Virchow, McKeown, Goldberger and others advocated and, in line with Chargaff’s vision of a more truth-seeking, non-establishment science, to turn our attention to the process of adverse development which may be the source of the increasing prevalence of AIDS diseases in the countries of Africa and other parts of the Third World.

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