Necessity and sufficiency in the aetiology of HIV/AIDS: The science, history and politics of the causal link

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Public debates on HIV/AIDS in South Africa have, for the last three years, been dominated by the controversy around the causal link between HIV and AIDS. A decision on this question has direct consequences for health policy, treatment, and education. However, this question also warrants a medical-historical investigation into concepts and models of causality and the way they have panned out in medical-scientific revolutions, and in diagnostics and treatment. As this paper attempts to show, necessary and sufficient criteria for disease causation are crucial in the debates on the aetiology of HIV/AIDS. In the course of the history of medical diagnostics, the sufficiency criterion has been considerably modified, while the necessity criterion has been foregrounded. It has been shown that the difficulties surrounding the establishment of strict sufficiency criteria do not preclude the elaboration of an aetiology of HIV/AIDS. While mainstream medical science privileges the necessity criterion, the AIDS dissidents insist on strict sufficiency for conclusive proof of the causal link between HIV and AIDS. This paper aims to show that both criteria have a role to play, but in differentiated ways and at different and distinct sites of intervention.

Key words: aetiology of HIV/AIDS, causality in medical diagnostics, necessary and sufficient causes, medical history

Introduction

In the last three years or so, public AIDS debates in South Africa, insofar as they reached a high profile and have commanded the attention of the media, have been dominated by the controversy around the causal link between HIV and AIDS. The statements by the State President in his debate with opposition leaders, and within the temporarily established Presidential AIDS Advisory Panel, have created further divisions between central government and provincial and local government health departments, between State Health and NGOs working in the HIV/AIDS arena, between people living with HIV/AIDS and local and national health structures, and between government officials and AIDS awareness educators and activists. Expressing an opinion for or against the causal link between HIV and AIDS has become not only a scientific, but also a political credo, with dramatic consequences for AIDS education,¹ policy, health care delivery and treatment. President Thabo Mbeki’s statements on the subject (most prominently in the article in *Time Magazine* and in his speech at the 13th International AIDS Conference in Durban in July 2000) have served to catapult the issue of the causal link between HIV and AIDS into the limelight.

In this paper, I would like to look at the question as to why and how the causal link between HIV and AIDS could become such a politicised issue. While the question of this link commands enormous public interest on account of its immediate consequences for health policy and treatment of HIV/AIDS, it also involves a medical-historical investigation. What is at issue in this broader picture, I would like to argue, is the concept of causality and the way it has panned out in the structure of scientific revolutions, and in medical diagnosis in particular. It will turn out that, beyond the immediate urgency of finding a cure for the disease that has been described as the most serious threat to all social arenas in Central and Southern African countries, there are wide-ranging issues at stake, including the origins of Western biomedicine, the very definition of scientific medicine, and the question of the relationship between politics and science/medicine more generally.

While I am amplifying the issue of the causal link between HIV and AIDS to look at the problem of causality in medical diagnosis and its history, I would like to narrow my task by adding a few disclaimers at the outset. I am not in the business of affirming or refuting the claims of AIDS revisionism and its South African derivatives. What I hope to show are the limitations in the way that the question as to whether or not HIV causes AIDS, has been posed. My critique to that effect is directed at both the AIDS revisionism lobby, and the way in which the South African media have stylised this issue into a political credo. The level of com-
plexity in the analysis of causal factors in the diagnosis of retrovirally determined diseases makes a mockery of the question, ‘do you or don’t you believe that HIV causes AIDS?’.

Causality in medical diagnosis

One of the foundational if not definitive epistemological ‘events’ for modern Western biomedicine is the establishment of specific criteria of causation for diagnostics. The same foundational event has, however, proved the Achilles heel of medicine from the end of the nineteenth to the end of the twentieth century. The theory of causes remains the weakest spot of pathology. Herein lies the truth of AIDS revisionism, quite in abstraction from its more specific claims. It is instructive to note that in trying to prove that the link that would establish a causal relation between HIV and AIDS is insufficient, Peter Duesberg (1987; 1989; 1994a; b) recurs on principles formulated at the inception of bacteriology in the second half of the nineteenth century. In order to demonstrate the status of this event in the history of medicine, as well as the loop of the return to the founding statements of bacteriology, I would like to unravel its implications, mutations and reformulations historically.

From the 1850’s onwards, the limitations of symptomatic notions and treatments of disease were recognised. Scepticism had been mounting over the legacy of the classificatory medicine of natural history of the 18th century, and over vague and imprecise claims about disease causation that referred any morbid alterations to a range of different unrelated and non-specifically sufficient causes, whereby an set of conditions was non-specifically related to the occurrence of a disease. A new generation of medical scientists and researchers claimed that not enough attention was being paid to the origins of the disease process. The urgent need to curb the incidence and prevalence of infectious diseases of both animals and humans spawned the quest for specific causes of specific diseases. In the process, the type and the very definition of aetiology underwent a substantial shift. While aetiologies — the assignment of causes to phenomena and processes of change in medicine and psychiatry — were around for as long as mankind has attempted to establish causes for diseases, medical-scientific researchers since the mid-nineteenth century broke with all previous notions of the relation between causative agents and disease, assuming that a single necessary cause must be identifiable for every case of a particular infectious disease. They thereby systematised non-specific notions of causes (e.g. notions of occult influence, or of cosmic forces acting on the organism), by borrowing from the philosophical elaboration of the laws of causality since Hume. As normative spheres were being increasingly differentiated in modernity, science could bracket the value attached to qualities of life, in order to attain knowledge of the processes of life. It should therefore come as no surprise that abstract laws of causality, as elaborated in the field of Newtonian physics and mechanics, found their application in scientific medicine in its early stages. Only through a de-naturalisation of nature, the de-materialisation of matter in physics and chemistry, and the de-vitalisation of life in biology, could functions and processes in the life sciences be analysed. A theoretical analysis of the natural world could only proceed on the basis of what was artificially induced (since the outgoing 19th century in the laboratory), to expose latent causal relations, with the aim of making life processes manifest (Canguilhem, 1979, pp. 153, 126).

The widening gulf separating scientific medicine based on deductive-nomological models, from ‘the art of healing’, from the experience of illness and disease, and from clinical praxis has been decried by its critics in terms of a ‘hiatus theoreticus’ (Paul, 1996). However, the postulated link between clinical methodology and scientific theory is partly re-established in scientific medicine by the aim of the cure, which reinstates the priority of necessary causes (which assert that for every case of a disease, a specific causative agent must be present, and that without the presence of that specific causative agent, the disease would not occur). The establishment of a necessary cause of disease directly implies a therapeutic orientation: ‘In clinical medicine, it is generally assumed that rejecting a true hypothesis (e.g. missing a diagnosis of acute appendicitis) is worse than accepting a false hypothesis (e.g. performing an unnecessary appendicectomy)’ (Fagot, 1984 p. 102). While monocausal aetiologies have been challenged if not rejected by medical scientists since the end of the 19th century, this does not affect the crucial role of necessary causes in specific diagnoses. Linear, monocausal accounts of disease causation have to some extent been modified by conditionalist accounts which explicitly or implicitly draw on John Stuart Mill’s theory of causality stated in his System of Logic (1843) (notably in Vol. I, Book 3, chs 4–6, and Vol. II, Book 3, ch. 21). In Mill’s account, a ‘cause’ holds between a number of antecedents and a consequent. In the denomination of the cause, one of the antecedents is being singled out among all others, which are thereby rendered mere conditions. Thus, the cause is a necessary condition, without which the consequent would not have occurred or existed, but which is not of itself sufficient to bring about the consequent (Parkinson, 1988 p. 284). This model becomes operative in medical diagnosis and treatment. In prioritising one cause among a number of antecedent conditions, physicians have to weigh up the costs of different ways of being wrong. Assigning a cause thus involves ethical decisions, as well as the criterion of cost-effectiveness: ‘One is attempting to find indications that will allow one to make a choice of a cause in a way that will be most cost-effective with regard to interests in avoiding morbidity, risks of mortality, as well as financial costs’ (Engelhardt, 1984, p. 181).

Koch’s postulates: Necessary and sufficient causes

The search for criteria for specific diagnosis and therapy of infectious diseases was to make for a paradigm shift in medicine. It was Koch’s discovery of the tuberculosis bacillus, presented in a paper (‘Die Aetiologie der Tuberkulose’: Koch, 1912), delivered in 1882, that definitively established the dominance of germ theory. This and subsequent papers on the aetiology of TB (‘Ueber die Aetiologie der Tuberkulose’, 1882 and 1884) were the culmination of the previous decade of Koch’s work on wound infection, recur-
rent fever, and anthrax, in which he had attempted to arrive at an explanation of specific and necessary disease causation. In the 1870s, under the impact of the Franco-Prussian War and the attendant medical problems for armies and individual soldiers, Klebs' and Koch's surveys on the role of bacteria in disease aetiology focused primarily on wound infections. Koch's work on the causes of infectious diseases was pivoted on the elaboration of a philosophical theory of causality that provided the basis for modern medicine. In his investigations between 1878 and 1884 on the aetiology of anthrax (two papers of 1878 and 1881), on wound infections (two papers of 1878) and certain other papers (those published between 1882 and 1884), Koch variously developed a combination of necessary and sufficient criteria for disease causation. Earlier accounts of infectious diseases had focused on miasma, humours, or any set of various unrelated causes, mounted as explanations of a set of symptoms. Even explanations originating from nineteenth century pathological anatomy and cellular pathology, while much more specific, implicitly relied on non-specific sufficient criteria. Koch's investigations, in contrast, went in search of necessary criteria of disease causation. In other words, (certainly in his two papers on anthrax) he was primarily interested in proving that the presence of certain bacilli is necessary for the occurrence of a specific disease. This proof was initially stated in terms of an absence argument: without the presence of certain bacilli, the specific disease does not occur. With the publication of his papers on wound infections in 1878, Koch refined his criteria for disease causation: conclusive proof of the parasitic origin of a certain disease 'would require that we find parasitic organisms in all cases of the disease, that they are present in such numbers and distribution that the disease symptoms can be explained, and that a morphologically distinguishable organism is identified for every different disease' (Robert Koch: Neue Untersuchungen über die Mikroorganismen bei infektösen Wundkrankheiten (1878). Quoted in: Codell Carter, 1985, p. 357). Later Koch realised that morphological distinctness of specific bacteria as the agent for a specific disease is not a sufficient distinguishing criterion: something more than morphological distinctness is required to account for the distinct effects that the bacteria have on the organism. Consequently, he insisted that 'every possible characteristic of different strains of organisms be considered before identifying them as of the same species' (Codell Carter, 1985, p. 358). In finetuning the conditions for the necessary criteria of causation, Koch stumbled on sufficient criteria for causation. However, he battled to elaborate the criteria that would satisfy strict sufficiency. He found, for instance, that the disease agent might be something other than the bacteria (as for instance, a substance associated with the bacteria); or that the agent of the disease could also occur non-pathogenically in other instances or in other hosts.

It was with his later work on the aetiology of TB that the elaboration of strict sufficiency criteria became a major preoccupation for Koch. He observed that establishing a regular coincidence between a disease and a specific organism does not prove causality, nor does the location of the bacteria in the organs where the disease is known to originate (Codell Carter, 1985, p. 360). He proceeded to follow in the footsteps of Edwin Klebs, who had stated in his 1872 paper on gunshot wounds ('Beiträge zur pathologischen Anatomie der Schusswunden'), that 'tracing the invasion and the course of the micro-organisms can make causality probable, but the crucial experiment is to isolate the efficient cause and allow it to operate on the organism' (Codell Carter, 1985, p. 365). In an 1875 paper ('Beiträge zur Kenntniss der Schistomyceten'), he called for producing experimental evidence to establish causal relations rather than simple co-incidence. Such evidence could be obtained, he claimed, by 'isolati[ing] substances from the body and us[ing] them to induce further cases of infection' (ibid.). Driven, in addition by his dispute with Pasteur, while following Pasteur's method, and under pressure to justify his findings and procedures to academic medicine and 'normal science' accustomed to the search for sufficient causes as the exclusive paradigm of diagnostics, Koch started to isolate the suspected disease agent in pure culture. To prove that the isolated bacterium is really the cause of the disease, it remained to be shown that animals inoculated with the pure culture would contract the particular disease in every instance of such inoculation (Codell Carter, 1985, p. 360). In the demand for such demonstration, Koch followed Pasteur's reasoning (in the latter's studies on the spoilage of beer, published in 1875) on the combination of necessary and sufficient causes for the occurrence of contamination.

The combination of necessary and sufficient criteria for disease causation has been summarised (on the basis of Koch's investigations and publications between 1878 and 1884) as Koch's Postulates as follows:

- An alien structure must be exhibited in all cases of the disease.
- The structure must be shown to be a living organism and must be distinguishable from all other micro-organisms.
- The distribution of micro-organisms must correlate with and explain the disease phenomena.
- The micro-organism must be cultivated outside the diseased animal and isolated from all disease products which could be causally significant.
- The pure isolated micro-organism must be inoculated into test animals and these animals must then display the same symptoms as the original diseased animal.

Postulates 1 and 2 and the corollary concern necessary criteria for causation of the form 'in all cases of a specific disease B, a specific agent A must be present and detectable'. It is the first three postulates — those that concern natural cases of disease — that Koch never abandoned. Postulates 4 and 5 — those that demand inoculation tests — concern sufficient criteria for causation of the form 'wherever a specific agent A is present and detectable, a specific disease B must occur'. Sufficiency proof by means of isolation in a pure culture is invoked only when it is to be established that some bacterium can by itself cause a disease.

**Viruses / bacteria in search of disease**

However, despite the development of new culture media (since 1882) that allowed for new techniques in pure culture, the sufficiency criteria elaborated in postulates 4 and 5
proved a stumbling block for bacteriology and for modern medicine's account for disease causation in general. Koch could not prove that usually pathogenic organisms could never occur non-pathogenically. He discovered the phenomenon of healthy carriers in about 1893 (Codell Carter, 1987, p. 88). The famous case in point is the one in which tubercle bacilli can exist in a host without causing the disease. Similarly, it turned out that only a fraction of people infected with the cholera bacterium actually get the disease.11 This possibility calls into question both sufficiency criteria listed as postulates 4 and 5. "...the disease could still be caused by a substance other than the organisms themselves and therefore, there might be no way of inoculating pure bacilli to produce the disease" (Codell Carter, 1985, p. 362). Formulated another way, the presence of bacteria is not sufficient to explain the onset of disease.

Similar reasons underlie the aspersions cast on Koch's claim of having discovered a specific remedy to target the specific agent for tuberculosis. This was concretely manifested in a number of hitches that the history of bacteriology has become (in)famous for. In October 1890, Koch spectacularly announced a specific remedy for tuberculosis, under the name of 'tuberculin'. This announcement was met with enthusiastic public applause; however, not three months had passed when it turned out that tuberculin did not produce the supposed specific effect. As the media scandal termed the 'tuberculin fraud' ('Tuberkulinschwindel') unravelled, it was revealed that Koch had failed to isolate a single substance responsible for the effects he observed (Gradmann, 1998).

This insight in retrospect gave some credibility to the challenges that cell pathologists and conditionists confronted bacteriology with and, even more remotely, to the self-experiments by which the anti-contagionists in the middle of the nineteenth century demonstrated that the presence of bacteria is not sufficient for infection to occur.12 With this insight, which the bacteriologists had to concede, the focus of aetiology in bacteriology and virology shifted to infection without disease or, metaphorically speaking, to 'viruses in search of disease'. In other words, the investigation of the responses of the healthy body, or the body that was infected but did not become symptomatic, became an integral part of bacteriological demonstration. This is what has come to characterise the science of epidemiology: mass screenings to determine the prevalence of latent infection in otherwise healthy individuals. (A case in point were mass screenings for syphilis, by which 'hidden cases' were to be brought to light — i.e. people who had contracted the disease without knowing it, or while denying it, or without showing any symptoms of it.) In this way, the crisis of bacteriology was resolved in part epidemiologically (rather than aetiologically),13 with wide-ranging implications for preventive and promotive health initiatives, understood within an integrated social policy framework of either fascist (eugenic) or socialist or welfare-state orientation.14

Closely implicated in the contestations over causality in diagnostics are the sciences of immunology and serology that made their breakthroughs in tandem with those of bacteriology. The most famous serological reaction — the Wassermann test — was hailed as fulfilling the condition of specificity that became the fundamental law of immunology and the precondition for immunodiagnostics. As Julius Citron, collaborator of Wassermann, explained in 1910: "[The fundamental law of immunology is that] every true antibody is specific and that all non-specific substances are not antibodies" (Löwy, 1993, p. 74). August Wassermann and his collaborators on the test, Albert Neisser and Carl Bruck, assumed that syphilis — especially in its late clinical manifestations such as tabs and dementia paralytica — could be diagnosed by the presence of specific treponema pallidum antibodies in the blood. It was found difficult to demonstrate the presence of such anti-bodies in the blood though, because treponema pallidum could not be grown in vitro. Researchers therefore resorted to the indirect method of complement fixation that reveals the presence of specific antibodies in the blood.15 Yet the aetiological link remained to be established. Aspersions were cast from a relatively early date (i.e. 1907–1908) on the assumed and claimed specificity of the Wassermann reaction, as it was found that sera from syphilitic patients reacted also with extracts of organs from non-infected individuals (Löwy, 1993, p. 76). However, as the technical aspects of the test were being perfected, the debate about the aetiological link between the Wassermann reaction and treponemal infection died down and 'the specific link between the Wassermann reaction and syphilis became one of the best established medical facts' (Löwy, 1993 p. 78). The initial assumption was that syphilis has a positive result on the Wassermann test (necessity) and that a positive result of a Wassermann test indicated the presence of syphilis (sufficiency). 'While a negative response to the Wassermann test was considered of doubtful diagnostic value, a positive response was seen as a solid proof of active syphilis' (Löwy, 1993). What is evident here is the reversal of the roles of necessary and sufficient criteria for causation. The necessary argument is rendered sufficient, and vice versa: the sufficient criterion assumes the place of the necessary one. A positive test result was seen as a near-absolute proof of syphilis, contributing to the widely held conviction of the high prevalence of the disease, especially among the urban poor (Löwy, 1993, p. 80). Only from the mid-1930's did doubts about the specificity of the Wassermann test re-surface. They were confirmed only after the Second World War, as a result of mass routine syphilis testing,17 and the elaboration of new methods of testing for syphilis which compared the non-treponemal, reagin-based syphilis tests and the treponemal tests based on the presence of specific antibodies. This combination of strategies for testing for syphilis revealed a high number of biologically false positives, i.e. a high percentage of people who have a high level of Wassermann reagins in their serum and who tested negative in treponemal tests. Correspondingly, interest shifted from the diagnosis of syphilis to the diagnosis of biologically false positives, which in itself became a new nosological entity.18 'A positive Wassermann test, once viewed as a manifestation of a specific disease, acquired in the 1950's and 60's the status [of] non-specific diagnostic indication...' (Löwy, 1993, p. 84).
Modification of Koch’s postulates: Aetiology without strict sufficiency

Koch’s major contribution in developing the aetiologies of infectious diseases had been the establishment of necessary criteria for the causation of specific diseases, in the interests of specific treatment and cure. While insisting on a combination of necessary and sufficient criteria for causation, and while attempting to arrive at strict sufficiency criteria in all his investigations, Koch himself found that in some cases — of typhoid fever, diphtheria, leprosy and cholera —, it was impossible to infect experimental animals with pure cultures and thereby conclusively demonstrate the sufficiency argument. In such cases, Koch did not hesitate to return to the necessity argument as the main criterion for conclusively establishing causality. In such cases, he believed he was justified ‘in stating that if...the regular and exclusive occurrence of the parasite is demonstrated, the causal relationship between parasite and disease is validly established’ (Robert Koch: Über bakteriologische Forschung (Address to the Tenth International Congress of Medicine in 1890). Quoted in Harden, 1992, p. 254). In histopathological studies where the sufficiency clauses of Koch’s postulates could not be met, likewise, researchers — one of the most prominent being Howard Taylor Ricketts (in 1909) — relied on demonstrating causal necessity, combined with indirect evidence in place of strict sufficiency criteria. In the face of increasing difficulties of demonstrating causal sufficiency in the mode advocated but never absolutely required by Koch, researchers tended to revert to Koch’s criteria for disease causation, stated in his papers on wound infections of 1878: conclusive proof of the parasitic origin of a certain disease ‘would require that we find parasitic organisms in all cases of the disease, that they are present in such numbers and distribution that the disease symptoms can be explained, and that a morphologically distinguishable organism is identified for every different disease’ (Robert Koch: Neue Untersuchungen über die Mikroorganismen bei infektiösen Wundkrankheiten (1878). Quoted in: Codell Carter, 1985, p. 357). In leaning on these requirements for demonstrating necessary causality, later researchers tended supplant the distinction between and combination of the necessary and sufficient criteria in Koch’s postulates with the distinction between necessary and incidental factors, mooted by Koch in his early elaborations.

This is particularly evident in the attempts to adapt Koch’s postulates to the science of virology, where the demonstration of sufficiency by isolating pure cultures and by inoculation is impossible10 (or, to be more precise, was impossible until the 1940’s which saw improved tissue culture techniques and immunological tests, as well as the introduction of electron microscopy). Convinced that the impossibility of detecting viruses in the tissue of the affected organism at all times and/or the impossibility of culturing viruses (e.g. in the case of hepatitis and infectious mononucleosis which eluded detection under the electron microscope and growth in cultures until the mid-1960s) did not invalidate them as aetiological agents, Thomas Rivers emphasised the requirement of specificity and necessity in his reformulation of Koch’s postulates for virology. For him, these two principles overruled Koch’s sufficiency criterion. He ‘emphasised that the polio virus was the cause of polio even if it could not be isolated from every case at any given time...’ (Harden, 1992, p. 263). According to Rivers, only two, but nonetheless demanding, rules of proof are needed to establish aetiology for viral diseases:

- A specific virus must be found associated with a disease with a degree of regularity.
- The virus must be shown to occur in the sick individual not as an incidental or accidental finding but as the cause of the disease under investigation. (Thomas Rivers: Viruses and Koch’s Postulates (1937). Quoted in Harden, 1992, pp. 256–257).

Robert J Huebner developed a nine-point programme by way of amplifying the guidelines laid down by Koch and Rivers for virology, aided by epidemiology, immunology and serology.20 In Huebner’s investigations, the criterion of specificity was modified, as ‘he recognised that several viruses might cause similar symptoms and that, for some illnesses, two or more viruses at once might be responsible’ (Harden, 1992, pp. 256–259). With this observation, any sufficiency proof would be rendered impossible. This, however, is not a failing of virological aetiology, but has been systematically elaborated by Alfred Evans (‘Causation and Disease: the Henle-Koch Postulates Revisited’, 1976) in an immunological account of aetiology, with simultaneously greater demands for specificity, for interdisciplinarity, and for conditionalism.21 In finding antibodies to specific antigens (and with the possibility offered by genetic engineering to clone an antigen to produce a vaccine which, if effective, would link the agent to the disease), immunology and serology have been particularly influential in linking an agent aetologically to a disease, even in the absence of virus or bacterium identification (Harden, 1992, p. 260). Thus the absence of antibodies need not preclude the possibility of an aetiology.22

In this sketchy historical overview over the procedures to prove disease causation, a number of salient summary points emerge. Firstly, the establishment of an aetiology is not precluded by the impossibility of demonstrating sufficiency; Koch’s investigations and procedures of demonstrating disease causation do, in fact, rely more consistently on necessary criteria, or a combination of necessary and weakly sufficient criteria23 for disease causation. Secondly, as Victoria Harden (1992) points out,

The central purpose of the various versions of Koch’s postulates, their modifications, and alternative criteria for establishing disease etiology has been to provide mechanisms by which the logical arguments of necessity and sufficiency could be satisfied. As sophistication in laboratory experimentation has increased, some particulars of these rules have changed. The characteristics of specific agents under investigation, furthermore, have also required alterations in method. Those investigators most successful in elucidating the etiology of new diseases have grasped the philosophical essence of Koch’s postulates and have shaped or reshaped their techniques toward solving a particular problem. (p. 263)

Solving a particular problem in turn, as I will attempt to show in what follows, is integrally linked to the identification
of necessary causes, as in fact US retrovirologists’ William Blattner’s and Robert Gallo’s arguments supporting HIV as the cause of AIDS are.

**AIDS revisionism: Return to sufficiency**

All the more puzzling seems Peter Duesberg’s insistence on those of Koch’s postulates that call for a strict sufficiency proof of causation, as an absolute requirement for the demonstration of a causal link between HIV and AIDS. Duesberg hereby implicitly relies on the elaboration of Koch’s postulates between 1878 and 1884 — the middle period of Koch’s investigations. This comes after Koch’s initial statement of the requirements of specificity and necessity, and before his expression of doubts about the possibility of meeting the sufficiency criteria in each and every case. In Duesberg’s understanding of Koch’s postulates, as expressed in his own re-statement of these postulates, he privileges the sufficiency argument (that with the organism present, the disease must occur, demonstrated through isolating the micro-organism from the host, growing it in pure culture, and having it reproduce the original disease when introduced into a susceptible host) over the necessity criterion (that without the organism, there is no disease: the micro-organism must be found in all cases of the disease).  

Duesberg’s return to this formulation of Koch’s postulates does not adequately take into account the modifications required for virology, immunology and serology, and their own demands for stringency. Harping on Koch’s postulates by relying on the fulfilment of sufficiency criteria in the explanation of disease causation is citing proof procedures that from the outset could not conclusively and universally establish proof, not even in the investigations of Koch himself. The difficulties in producing sufficient proof are compounded in the case of viruses, which are notoriously difficult to isolate directly; they could only be identified through antibodies as viral markers, and through other highly sensitive methods.

Duesberg’s arguments have been challenged by Blattner, Gallo and Temin who lay the foundation for the necessity argument, combined with the exclusion of accidental factors, as proof for the causal link between HIV and AIDS. Referring to the predictable sequence of seroconversion, progressive immunodeficiency and clinical AIDS; to vertical transmission; and to AIDS following transfusions of HIV-infected blood (which, when screened for HIV antibodies and excluded if positive, arrested the further incidence of AIDS in blood transfusion), they conclude ‘that a virus causes a disease if the virus is consistently associated with the disease and if disruption of transmission of the virus prevents occurrence of the disease’ (quoted in: Harden, 1992, p. 267).

An additional factor has been added to question the absoluteness of the sufficiency criteria laid down in Koch’s postulates. In his anthrax papers, as well as in his investigations into wound infections, Koch’s development of sufficiency criteria for proof of disease causation relies on inoculation experiments, which cannot automatically be extended to the claim that the organisms in question are the agents of the disease as it would occur ‘naturally’. Secondly, in attempting to lay down sufficiency criteria for the demonstration of disease causation, Koch relied to some extent (in the case of TB and wound infection, for instance) on animal inoculation experiments. In sticking to the letter of Koch’s postulates, Duesberg attempts to refute the causal link between HIV and AIDS on the grounds of the alleged failure to meet the sufficiency criteria, spelt out by Koch in relation to inoculation and animal experiments. For Duesberg, sufficient causation could be demonstrated if chimpanzees inoculated with HIV since 1983 would have developed AIDS (1994a, p. 2; 1994b, p. 4). However, the results of animal experiments can be extended to human infections only by analogy (Codell Carter 1985, p. 359). Such an analogy might be said to be stretched beyond its bounds in cases of viruses, as ‘most viruses are species-specific in host range and in capacity to produce disease’ (William Blattner, Robert Gallo, and Howard Temin responding to Duesberg, 1988, quoted in Harden, 1992, p. 266). Robert Gallo (1991, p.280) explains,

> It is often impossible to find an animal that a given human virus can infect..... Many viruses do not produce disease even when introduced into an animal; in many instances this is because they cannot under any circumstances infect the animal.

In insisting, in an *ex negativo* demonstration, on the fulfilment of sufficiency criteria in the establishment of a causal link between HIV and AIDS, Duesberg might be seen to open up a *hiatus theoretics* of a special kind, namely one between a precisely stated law of causality, and an etiology involving the privileging of one necessary antecedent condition among other variables, according to any one or combination of the more pragmatic considerations of therapy, prevention, ethical considerations, and cost-effectiveness, which might compromise strict sufficiency. Framed in terms of this gap, Duesberg’s challenge to the mainstream view might be seen to emanate from the precept of the non-reducibility of causality to etiology. This charitable interpretation of Duesberg’s attempts at disputing the causal link between HIV and AIDS, however, is unwarranted in the light of the consideration that he does not concede the necessity criterion for establishing causation, thus ruling out any stringent criteria of causation even on logical grounds.

Duesberg largely ignores the innovation of Koch’s postulates in the form of the requirement of necessary criteria for the explanation of disease causation. Instead, he pins his criticisms of mainstream AIDS research, treatment, and activism almost exclusively on the sufficiency criteria whose incompleteness he takes as evidence for the alleged invalidity of the causal link between HIV and AIDS. He takes his doubts about the fulfilment of the necessary criteria for disease causation so far as to advise to concentrate AIDS prevention efforts on AIDS risks, rather than on transmission of HIV. In thus insisting on non-specific sufficient criteria for disease causation (Duesberg, 1989, p. 16), he goes back behind Koch’s postulates.

**AIDS is not generally recognised as an infectious disease by the revisionists.** Duesberg proposes that HIV is a passenger virus. Antibody-positivity could, Duesberg suggests, indicate a retrovirus that persists as latent, non-pathogenic infection. While AIDS in America and Europe is said to be a non-infectious disease syndrome induced by acute
viral or microbial infections or drugs (either nitrite inhalants and other recreational drugs, or AZT and other drugs that attack the DNA chains in the cell), African AIDS has been proposed to be an unrelated epidemic caused by malnutrition, parasitic infections, and poor sanitation (Duesberg, 1989; Duesberg, 1994b).

It has been acknowledged by the critics of the AIDS dissenters, that in the mid- to late 1980s, Duesberg did ask the scientific community some valid questions about AIDS (see e.g. Hooper, 2000 p. 168). However, some of the central questions have now been answered, despite Duesberg’s continuing skepticism. Some of the weakest points remain Duesberg’s pronouncements on ‘African AIDS’, aspects of which we can trace in some of the official pronouncements of the South African government’s Department of Health and in the presidential pronouncements and policies. The specific case for ‘African AIDS’ and the notion of AIDS as a disease of poverty evidently has appeal in these circles on account of a stereotypical casting of risk groups (homosexual men and drug users) from which Africans are being exempted in Duesberg’s schema, and on account of the South African government’s developmentalist agenda (pursued under the motto of ‘poverty relief’). What might play into this appeal that AIDS revisionism holds, are reservations against some of the fundamental tenets of Western biomedicine.

The attributions to ‘African AIDS’ mark a return, full circle, to a non-specific sufficiency argument for disease causation before the formulation of Koch’s postulates in the 1870’s to 1880’s. To say that African AIDS is caused by poverty is to basically assert two different and contradictory propositions:

• There is no aetiology of AIDS that could be derived from a causal link between HIV and AIDS

• The cause for AIDS is poverty (This assertion is inherently self-contradictory: it fails to establish a specific causative agent, and thus it cannot act as a causal explanation.)

The first proposition asserts that there is no identifiable cause of AIDS, whereas the second proposition instates a non-specific sufficient criterion as the sole criterion of disease causation. In that sense, it is not unlike the correlation mentioned earlier, drawn between poverty and syphilis before 1940. However, the difference lies in the fact that even with the mistaken assumption of the specificity of the Wassermann test, the necessary criterion for disease causation was being upheld. In the case of the assertion that poverty causes AIDS, the onus of both necessary and sufficient proof procedures is thrown overboard, and diagnosis is being returned to a pre-scientific mode.

Disease and poverty, and the politics of medical intervention

This does not, however, mean to say that poverty has nothing to do with disease. The assertion of the link between disease/ill health and poverty, though not intrinsically progressive (it has its place even in eugenics programmes of totalitarian regimes), has been the hallmark of progressive health analysts and advocates of primary health or national health services in South Africa. The very notion of ‘diseases of poverty’, among which many preventable infectious diseases are listed, has featured as a critical term in the analyses and advocacies lobbying for primary and preventive social health programmes.

Until the mid-nineteenth century (i.e. before the postulation of necessary causes for diagnosis and treatment of specific diseases), physicians tended to be politically active in their self-appointed roles as social reformers (thus for instance, Rudolf Virchow). Their understanding of the mission of medicine was to actively intervene in political and social life. This view corresponded with the then dominant notion of non-necessary causation of ill health. In this view, social misery ranked high among the conditions of disease (Schlich, 1996).

Robert Koch and his collaborators (the most vociferous among them on this issue was Emil von Behring), and Theodor Kocher, in contrast, concentrated their interventions in the sphere of specific diagnosis and therapy, in which they developed specialised expert knowledge. If they were ostensibly ‘apolitical’, it is because of their different approach to the analysis of disease causation. The diagnoses and therapies developed by modern Western biomedicine are not therefore to be cast as being of ‘devil’s stuff’ (as they were initially greeted by academic and practical medicine versed in symptomatic or clinical diagnoses relying on non-specific sufficiency criteria). In this respect, it is worth considering the theoretical revolutions in medicine of the last hundred years, some of which have simultaneously revolutionised social medicine. This is how Koch (1912), for instance, announced his discovery of the causative agent of tuberculosis (in 1882) through the adherence to the principles of demonstrating necessary criteria for disease causation:

‘Until now, people were used to looking upon tuberculosis as expression of social misery, and hoped for a decrease in the incidence of the disease with the improvement of social conditions. That is why public health does not as yet know any measures directed against tuberculosis itself. But in future, the battle against this terrible scourge will no longer deal with a putative indeterminate something, but with a tangible parasite.’ (p. 444)

The innovation that bacteriology brought to the view of infectious diseases consisted in the identification and diagnosis of infectious and communicable diseases on the basis of a correlation of the disease in question with specific microbe agency. Individual conduct and social conditions, as well as climate, geographical location, and other conditional factors were thereby rendered secondary. ‘The identification of the specific germ of a disease allowed an individualisation of those forms of illness that were publicly regarded as scandalous and indicated purely medical ways of dealing with them. Once and for all, health had now been deprived of any religious, moral, … or philanthropic connotation…’ (Labisch, 1992, p. 95). For the individual patient, this potentially meant a liberation from stigmatisation, and from a sense of fear, shame and guilt.

Bacteriological therapies relying on identifying and targeting specific causative agents did not by their very definition remain confined to the individual in the privatised
patient-doctor relationship. They were integrated in public health programmes, e.g. sanitation geared to eliminate and prevent transmission through the specific causative agent (e.g. the purification of drinking water to prevent the outbreaks of cholera and typhoid), disinfection, and active and passive immunisation. It was, in fact, with the provision of universal free treatment for syphilis — in the form of Salvarsan — in the 1910s in Britain, that a precedent was being set for the integration of prevention and treatment in the public health service (Ross & Tomkins, 1997).

Thus, the simplistic valorised distinction between progressive preventive/promotive/primary health care delivery (being oriented towards an integrative holistic social programme), and conservative curative (tertiary or private) care (being individualised and de-politicised), cannot be said to hold.

Necessary criteria for the explanation and of disease causation are crucial in developing diagnostic techniques and therapies for the prevention and control of infectious diseases. The supposition that there is no disease without the presence of an organism specifically linked to the disease, implies a therapeutic orientation from the outset: the elimination of the organism entails the remission or cure of the disease. Thus, diagnosis, prevention and treatment require the fulfilment of the necessary condition for disease causation. It is not co- incidental that nineteenth century critics of pathological anatomy and cellular pathology accused their forebears in academic medicine — notably Claude Bernard and Rudolf Virchow — of not having made any contribution to the development of cures for common diseases. Edwin Klebs, for instance, observes in his 1877 and 1878 lectures that while pathological anatomy had made great strides in describing and explaining disease processes, it had contributed almost nothing to the control of diseases. This, he recognised, was due to the pathologists’ peculiar conception of disease causality, which was confined to non-specific sufficient causes (Codell Carter, 1985).

Klebs’ formulation of the merits and de-merits of pathological anatomy contains clues for a possible evaluation of the requirements of causal explanations in medical diagnostics. In his statements, the identification of necessary causes is instrumental to the control and cure of infectious diseases that marks the achievements of modern medicine. That does not, however, rule out the role of sufficiency arguments. While sufficient causes are virtually irrelevant when it comes to controlling diseases, ‘an interest in sufficient causes makes perfect sense on the assumption that medicine is pre-eminently a mechanism for reinforcing social norms’ (Codell Carter, 1991, p. 548). Sufficient causes are also validly invoked in attempts to explain why something happens in a particular case.

As Codell Carter explains, it is the context of an investigation that determines whether one seeks a sufficient or a necessary cause (1987, p. 88). However, for a comprehensive strategy on HIV/AIDS, it will not do to simply relativise these two broadly defined positions. Even for AIDS education, explanation by reference to non-specific sufficient causes will not suffice. And conversely, for the prevention of mother-to-child transmission, the simple administration of a dose of nevirapine will not do. The combination of the two types of intervention based on an analysis of necessary and weakly sufficient causes, has, for instance, been made mandatory in testing and counselling — there should be no testing without counselling (even with the availability of rapid tests). However, the two types of intervention need to be distinguished to prevent a confusion whereby explanation by reference to non-specific sufficient causes replaces the identification of a necessary cause.

Notes

1 As, for instance, those recorded by a HIV/AIDS counsellor, Florence Ngobeni: she states that the number of people coming to be tested in Chris Hani Baragwanath Hospital’s perinatal HIV research unit is dwindling, as many of them believe that they are not at risk. Also declining is the number of couples who come for counselling — with many men refusing to attend counselling sessions. Furthermore, in her opinion, the poor compliance with treatment regimens is partly due to the confusion generated by the mixed messages received by people on HIV/AIDS since the 13th International AIDS Conference in Durban in July 2000. She expresses concern that the mixed signals being sent out by the government are slowly eroding [the] gains in the way of educating people, particularly women, about protecting themselves from the virus — and promoting healthy living for those already HIV-positive’ (Magardie, 2000, p. 4).

2 The problems that early bacteriologists address themselves to, give an indication of the urgency to find ways of controlling diseases prevalent in the nineteenth century: chilbed fever, cholera, tuberculosis, diphtheria, diseases killing silkworms, diseases of cattle and sheep (notably anthrax), and also malaria.

3 Hume’s third criterion (elaborated in the Treatise of human nature (1739)) for the relation between cause and effect stipulates a necessary connection, by which it must be true to say that if whatever is identified as the cause had not been, whatever is identified as the effect could not have occurred. Causal necessity, for Hume, involves a constant conjunction of what is identified as cause, with what is identified as effect.

4 An instructive example of such de-naturalisation in scientific medicine is provided by Canguilhem in referring to the work of Paul Ehrlich: Ehrlich’s contribution was the systematic utilisation of affinities of chemical dyes for the production of artificial antigens. This involved the construction of a molecule — quite far removed from its practical application (Canguilhem, 1979, p. 124).

5 Another factor enhancing the prestige and public standing of bacteriology could be seen in the bacteriological diagnosis and treatment of patients during the Hamburg cholera epidemic in 1892 (Gradmann, 1998).

6 Koch’s postulates owe a great deal to Hume’s third criterion (elaborated in the Treatise of human nature (1739)) for the relation between cause and effect: namely that of a necessary connection, by which it must be true to say that if whatever is identified as the cause had not been, whatever is identified as the effect could not have occurred. Causal necessity also, for Hume, involves a constant conjunction of what is identified as cause, with what is identified as effect.

In a more recent study of the concept of cause (one that postdates Koch’s postulates, though), RG Collingwood identifies the cause of the event in the life sciences as something by means of which we can control that which is caused: ‘The cause of an event is the handle, so to speak, by which human beings can manipulate it… [A cause is] an event or state of things which it is in our power to produce or prevent that whose cause it is said to be’ (Collingwood, 1940, pp. 296–297). The latter statement of the concept of cause is one that best describes the scientific revolution
that bacteriology brought to the history of medicine.

The uneven nature of his papers, and of the stipulated combination of causes is documented in an excellent paper by Codell Carter (1985): *Koch’s Postulates in Relation to the Work of Jacob Henle and Edwin Klebs*.

Such as those listed in French encyclopaedic dictionaries of medicine of the middle of the nineteenth century, as quoted by Codell Carter: Diabetes, for instance was stated as having been observed ‘in subjects habitually exposed to a humid, frigid, and somber atmosphere, and who lead a sedentary life, in those who abuse aperos, diuretic, fermented, or alcoholic drinks, who are given over to excessive onanism, or venery, or whose constitution has been weakened by influences of every kind.’ In the same work, Codell Carter (1991, pp. 537–538) notes, ‘epilepsy was ascribed to fear, anger, violent emotions, excessive venery, habitual onanism, the sight of epileptics, the abuse of alcoholic drinks, the consumption of lead, and worms in the digestive tract.’

Ophthalmia was said ‘to include the unexpected suppression of perspiration or of a habitual secretion such as a haemorrhage from the nose or anus; repercussion of scabbing of the skin or of a wound; scrophulous or syphilitic vice; and cold and humid air’.

Examples abound.

The gist of Pasteur’s experiments have been summarised by Codell Carter (1991, p. 531) in terms of two main lines of argument: ‘In the first, [Pasteur] contended that ordinary airborne dust contained particles indistinguishable from the germs of living organisms. When such dust was introduced into fermentable liquids, colonies of living organisms soon flourished there, whereas the same liquids remained pure if they were exposed to dust or to ordinary air that had been heated sufficiently to destroy living germs’.

Pasteur had criticised the notion that the presence of spores was a necessary cause of anthrax, pointing out that the privileging of necessary causative criteria did not take sufficient account of sufficient causes. While Pasteur found germs necessary for the generation of cultures, sufficient causes for the generation of cultures were dependent on suitable background conditions (see Carter, 1991, p. 532). Pasteur furthermore tried to establish sufficiency proof through pure culture and inoculation in both his experiments with ammoniacal urine and with one of the causative agents of a specific silkworm disease (*flacherie*).

Robert Gallo (1991, p. 279) extends the latter case into the following generalisation: ‘...subclinically infected — and totally asymptomatic — carriers are the rule. And this is not an aberration restricted to cholera. Expression of disease is the exception for the majority of microbes.’

One of the most famous of these self-experiments is the one by which the anti-contagionist Ludwig von Pettenkofer wanted to demonstrate that the comma bacillus is not sufficient reason for infection with cholera. At a conference, he dramatically drank cholera vibrons, without succumbing to the disease.

This was the subject of a lecture by J Andrew Mendelsohn: Von der Beweiskraft zur Krise: Ätiologie der Infektionskrankheiten 1890–1910, held on 18 June 1996 at the Institute for the History of Medicine of Humboldt University, Berlin.

Epidemiology has been defined as the study of disease occurrence in human populations, with the aim of preventing disease occurrence by intervening against factors causing disease in the population. It is understood that the latter are encapsulated in the notion of a web of causation consisting of causal chains. This notion has become instrumental in prevention strategies, ‘since blocking the causal role of but one component of a sufficient cause renders the joint action of the other components insufficient and prevents the effect’ (Norell, 1984, p. 133).

Epidemiological research focuses not just on necessary and sufficient causes for the occurrence of a disease, but includes in its frame of reference contributing and component factors, such as relative risk, exposure, and furthermore, factors like pollution, smoking, diet, immunisation, population density, climate and fauna. It establishes the degree of causal influence through a correlation analysis. ‘The relevant measure of disease occurrence...[is] a measure of incidence, such as the proportion of individuals falling ill during a certain period of time (the Cumulative Incidence Rate) or the number of cases per person-year at risk (the Incidence Rate).’ (Norell, 1984, p. 129).

This complement fixation test is described as follows: ‘*The complement fixation test is based on the principle that when antibody-containing serum is allowed to react with a specific antigen (in the case of the Wassermann test, an extract of a syphilitic liver, rich in treponema antigens), in the presence of guinea-pig complement, the complement will be absorbed by the antigen-antibody complexes. This disappearance of the complement from the reaction mixture can then be demonstrated by a revealing system.*’ (Löwy, 1993, p. 76).

The association between syphilis and poverty was one of the assumptions on which the infamous Tuskegee Syphilis Study, conducted by the Public Health Service over a 40 year period (starting in 1932), was based. This association was compounded by racist stereotypes. The mindmaster of the study believed that virtually all Southern blacks were infected, and that the men involved would never be treated anyway. ‘Over a forty-year period, the Public Health Service actively sought to prevent the men from receiving therapy, all the while telling the subjects that they were being treated by the government doctors. Many of the men — perhaps more than 100 — died as a result of tertiary syphilis.’ (Brandt, 1985, p.158).

Here again we can see the shift to the examination of the healthy organism as important component of bacteriological diagnostics.

For instance, a chronic biologically false positive diagnosis in young women was taken to indicate the earliest sign of a severe auto-immune disorder — e.g. lupus erythematosus). Other cases of chronic biologically false positives were linked with a vast array of chronic systemic disorders, or with substance abuse of hypertension drugs (Löwy, 1993, pp. 84–85).

While cultivation of viruses in tissue culture is a process similar to cultivation of bacteria in lifeless media, not all viruses can be cultured (Thomas Rivers: Viruses and Koch’s Postulates. In: Harden, 1992, p. 257), and certainly not outside of cells. Robert Gallo (1991, p. 280) explains: ‘It is often impossible to find an animal that a given human virus can infect. ... Many viruses do not produce disease even when introduced into an animal; in many instances this is because they cannot under any circumstances infect the animal.’

Huebner’s guidelines have been paraphrased as follows:

- The virus must be established as a real entity that can be cultured in other laboratories.
- The virus must be shown to be of human origin and not a contaminant or a virus from experimental animals.
- The virus must be shown to produce an active infection by evoking an increase in serologically demonstrable antibodies.
- The virus should be characterised early, so that comparisons can be made as promptly as possible with other agents already described or soon to be discovered.
- The virus should be constantly associated with a specific illness.
- Double-blind studies of the virus in human volunteers should be made, with proper adjustments for the subjective impressions produced in both observers and subjects. This is of the utmost importance when studying poorly defined illnesses that are, nevertheless, familiar to all.
- Carefully conceived epidemiologic studies co-ordinated with adequate laboratory and clinical observations are indispensable for the purpose of finally establishing the etiologic role of highly preva-
lent viruses in human disease. Two types of studies are generally employed:

- Studies of populations experiencing a disease outbreak;
- Long term studies of community or institutional groups.

• Prevention by specific vaccination. If a vaccine prepared from a suspect virus prevents a specific disease, the virus may be said to cause the disease.


21Evan's 'Five Realities of Acute Respiratory Disease' are listed as follows:

• The same clinical syndrome may be produced by a variety of agents.

• The same etiologic agent may produce a variety of clinical syndromes.

• The predominating agent in a given clinical syndrome may vary according to the age group involved, the year, the geographic location, and the type of population (military or civilian).

• Diagnosis of the etiologic agent is frequently impossible on the basis of the clinical findings alone.

• The cause (or causes) of a large percentage of common infectious disease syndromes is still unknown.' (Alfred S Evans: Causation and Disease: The Henle-Koch Postulates Revisited (1976). Quoted in Harden, 1992, p. 259, n. 29).

22This observation would take care of Duesberg's contention that many cases of AIDS without HIV have been documented (1994a, p. 3; 1994b, p. 1).

23Weakly sufficient criteria are defined as those which are methodologically precarious, and only weakly, indirectly, and incompletely confirmable. This does not render them meaningless, though: they can play a role in explanation, and in answers to 'iffy' questions (contrary-to-fact conditionals) (Feigl, 1953, p. 410). In aetiological accounts, weak sufficiency is predicated on one or more conditions that relate to, but fail short of logically strict sufficiency.

24Duesberg re-states Koch's postulates as follows:

• The micro-organism must be found in all cases of the disease.

• It must be isolated from the host and grown in pure culture.

• It must reproduce the original disease when introduced into a susceptible host.

• It must be found present in the experimental host so infected.' (Retroviruses as Carcinogens and Pathogens: Expectations and Reality (1987). Quoted in Harden, 1992, p. 264).

Duesberg's insistence on the fulfilment of strict sufficiency conditions might be motivated by an analysis of the history of mis-diagnoses. He makes the lack of attention to the elaboration of the sufficiency argument, or the abandonment of Koch's postulates tout court responsible for the mis-diagnosis of the Epstein-Barr virus as the cause of Burkitt lymphoma, until Burkitt lymphomas free of the virus were discovered (Duesberg, 1989). On the same grounds, he moots the possibility of a high number of false positives in the diagnosis of AIDS by antibody presence (Duesberg, 1987).

25It has, for instance, not been possible to produce infection in animals after introducing EBV, hepatitis B and influenza.

26The hostility which initially greeted bacteriology was exemplified by the reception of the bacteriological diagnosis of TB in Japan, where symptomatic diagnosis remained the preferred method for a while to come, as the identification of a necessary cause was feared to expose the patient to traditional anxiety and stigmatisation (William Johnston: A Genealogy of Tubercular Disease in Japan. In: Social History of Medicine 7 (1994), pp. 246–247; cited in Schlich, 1996, p. 222).

In Germany and the United States, the chemotherapeutic cure for syphilis promised by Paul Ehrlich and Sahachiro Hata's announcement of Preparation 606 — an arsenic compound —, also named Salvarsan, (or, as it became popularly known by Ehrlich's own description of its therapeutic effects, 'the magic bullet'), made publicly available in 1910, was initially greeted with enthusiasm. However, it evoked a scandal on the basis of Paul Ehrlich's claim of a novel therapeutic procedure that could target and destroy the specific micro-organisms responsible for syphilis, bringing an immediate cure through a single injection (the latter of which proved elusive, as it was found that in most cases, a single dose was not sufficient to eradicate the spirochaetes completely). In both Germany and Britain, a lobby of medical, official and lay opinion claimed that offering this treatment was tantamount to encouraging sin and vice. Occasioned by the establishment of the Royal Commission on Venereal Disease, a high-profile debate erupted between the moralists advocating education for chastity, and the medicalists arguing for chemotherapeutic intervention for the sake of cure as well as prevention. After initially opposing the prohibition of Salvarsan and the regulation of its administration, health legislators responded positively to popular demands for the regulation of the production and administration of Salvarsan (Sauerteig, 1996). In Germany and in Britain, 'the promise of a cure for syphilis ... was only the beginning. Questions of how the drug was to be produced, distributed, and administered, and the respective roles of the state, the pharmaceutical industry, and medical practitioners loomed large' (Roes & Tomkins, 1997, p. 403). In both German and British public opinion, Ehrlich's patent and the manufacturer Hoechst stood accused of putting profit motives first.

In the United States, it was not until the 1930's that the social and economic costs of not dealing with the problems posed by sexually transmitted diseases, were being made explicit, thus weakening the moralists' view that had largely held sway. Until then, syphilophobia was widely held to be a deterrent more effective than prevention of infection. Prevention was supposed to be achieved through sexual abstinence. Chemical prophylaxis was seldom advocated. As in Britain, an acrimonious public debate erupted between the moralists and those dedicated to the new 'scientific' vision. However, the positions were often not as clearcut as all this. 'The debate between moral and scientific contingents... reflected a continuum of opinion, with doctors and health workers often sharing the precepts of those demanding sexual control, in spite of their attempts to combat the diseases through medical means' (Brandt, 1985, pp. 158, 46–47).

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