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DISSENT IN SCIENCE: STYLES OF SCIENTIFIC PRACTICE AND THE CONTROVERSY OVER THE CAUSE OF AIDS

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Abstract—In this paper, we use a scientific controversy, and the efforts to legitimize and undermine a theory, to examine the co-production of facts and the rules for verifying facts over time. We discuss these processes in terms of what we call 'styles of scientific practice.' In contrast to the focus of idealist philosophers on theory production and validation as forms of logic or ways of thinking, our styles of practice also include the activities of hands and eyes and the discourses between multiple actors in diverse situations. We discuss aspects of the different styles of practice deployed by opponents in a current controversy surrounding the etiology of AIDS to understand how the same data are interpreted in different ways to support diametrically opposed views. Our study describes and examines rules of confirmation used by supporters of the theory that HIV causes AIDS. For example, we introduce an 'epidemiological' style of practice used by AIDS researchers to synthesize information to understand this disease. Styles of practice stress the historically located collective efforts of scientists, technicians, administrators, institutions, and various 'publics' as they build and sustain ways of knowing. Yet, we also show that the 'history' is also a contested construction, not a given in dusty archives. We describe the different versions of history constructed by various participants in the debate to validate their current constructions and definitions of the disease AIDS. Finally, we discuss the politics behind disease definitions and the consequences of different definitions.

We may find it less instructive to determine whether a given account is true or false than to identify the diverse rules and conventions that govern whether and where a particular account is received as true or false, by whom, and with what material consequences [1].

INTRODUCTION: CONSTRUCTING FACTS WITHIN STYLES OF PRACTICE

Recent work in the sociology of science argues that the establishment of facts is a political process, with politics defined here as the efforts of scientists to empower and establish their own theories while simultaneously ignoring, de-legitimizing, and undermining the scientific arguments of opponents [2–4]. Many studies have examined scientific controversies in order to 'reveal' the politics of making facts [5]. This paper takes a slightly different angle on the politics of science. We use a scientific controversy, and the efforts to legitimize and undermine a theory, to examine the co-production of facts and the rules for verifying facts over time. We discuss these processes in terms of what we call 'styles of scientific practice.'

The construction and verification of scientific theories are *social* activities [6]. Styles of practice are historically located and collectively produced work processes, methods and rules for constructing data and theories and for verifying theories. We emphasize that theories are verified within styles of scientific practice. That is, statements, theories, or facts are 'true to' a set of self-authenticating techniques based within particular styles of scientific practice [7]. Thus, the practices (and rules) of theory verification are situated actions amenable to study by sociologists.

In this paper, we discuss aspects of the different styles of practice constructed and deployed by opponents in a controversy surrounding the etiology of AIDS to understand how the same data are interpreted in different ways to support diametrically opposed views. We also introduce a style of practice that we call 'epidemiological' which is used by AIDS researchers to confirm the theory that HIV causes AIDS. We give an account of the co-construction of this theory and the rules and practices for its verification.

Our approach to developing an understanding of the epidemiological practices is to examine a current controversy in AIDS research about the theory that HIV causes AIDS. We use a dissenter's arguments and the responses of scientists to his arguments to enter into the realm of scientific practice. Sociologist of science Bruno Latour has introduced a hypothetical imaginary dissenter in his book on *Science in Action* to challenge the scientist's portrayal of scientific facts as a method for opening up the arcane worlds of science to the novice. "To start our enquiry, we are going to begin from the simplest of all possible situations: when someone utters a statement, what happens when the others believe it or don't believe it. Starting from this most general situation, we will be gradually led to more particular settings" [3, p. 21]. In our case, a 'natural' dissenter has been provided to us, and his challenges to the HIV-AIDS research

eventually forced HIV-AIDS researchers to make explicit the practices behind their official positions [8]. We use these practices to construct our notion of an epidemiological style of practice.

In contrast to Latour's novice dissenter, our dissenter is a credentialed scientist who has challenged AIDS researchers in the language of retrovirology, epidemiology, and economics. His challenge has opened the scientific controversy to other public interested in AIDS research [9]. We follow the arguments and evidence appealed to by Peter Duesberg, a noted retrovirologist at the University of California, Berkeley, and a member of the National Academy of Science [10]. Duesberg challenged the theory that HIV causes AIDS, a theory that became widely accepted in 1984. Since that time, almost all AIDS researchers have accepted the theory and framed their research around this theme, although the exact mode of causation is still not understood [11]. In Duesberg's view, however, "science is still in action," and the issue is not settled. Duesberg examined and criticized the evidence proffered by the proponents and supporters of the theory. After every effort of HIV-AIDS researchers to silence his dissent and close the controversy, Duesberg opened it yet again, often with fanfare from alternative presses like *Spin* magazine and newspapers in gay communities. Duesberg's arguments also received attention from scientific journals such as *Nature* to the dismay of AIDS researchers who sincerely believe that Duesberg's views are harmful. In mid-1992 more mainstream media such as the *London Times* publicized Duesberg's views. At a special international symposium entitled 'AIDS, an alternative view' organized by the Foundation for Alternative AIDS Research and held in Amsterdam in August 1992, Duesberg's views shared the forum with recent work by Montagnier on possible co-factors and with AIDS researchers supporting the HIV hypothesis [12]. In late 1992 the *San Francisco Chronicle* and the *New York Times* published articles in their editorial pages for and against Duesberg's critique.

While Duesberg and some of his supporter portray him as a 'David' fighting against the 'Goliath' of the HIV-AIDS 'establishment,' individuals on the other side of the debate portray him as someone tilting at windmills. Each side accuses the other of illegal or immoral behavior. Instead of discussing this case in terms of personal psychology or self-interested behavior, we analyze it in terms introduced by the sociology, philosophy, and history of science.

As is the case with much recent work in the new social studies of science, we focus on the practices that create scientific facts, theories, and artifacts. In our account, what is being contested (and thereby explicated) in the debate is the history of the practice as well as what counts as verification. In the next section, we present a brief summary of the AIDS controversy. We then discuss the arguments in the debate in terms of styles of (scientific) practice and theory vindication. We give an account of how, in our case, different 'styles of scientific practice' support different means of verification. Our paper also presents an account of the construction of a complex new syndrome and the construction (and reconstruction) of the practices and rules for establishing disease causation in what we call the epidemiological style of practice [13].

A CONTROVERSY OVER AIDS ETIOLOGY

In April 1984 the United States Secretary of the U.S. Department of Health and Human Services Margaret Heckler proclaimed the first major victory in the battle against a horrible, new disease. She announced that a virus called Human T-cell Leukemia Virus-III or HTLV-III (later renamed Human Immunodeficiency Virus (HIV)) caused AIDS [14, 15]. Her announcement was based on the research of Robert Gallo, a noted retrovirologist at the National Institutes of Health, who had successfully isolated the virus [16]. Soon after the announcement, HTLV-III (now HIV) became the sole focus of research efforts against the disease. Despite the almost immediate and universal acceptance of the HIV-AIDS theory, Peter Duesberg, an eminent retrovirologist at the University of California, Berkeley, noted for his work on viral oncogenes, challenged the theory's validity. On the basis of his reading of the data available in the literature on AIDS, Duesberg concluded that HIV did not cause AIDS.

In 1987 Duesberg first publicly announced his conclusions in a review article in *Cancer Research* [17]. Based on his experience in retrovirology, he began by arguing that the basic characteristics of retroviruses were inconsistent with the etiology of AIDS. The article then examined the serological and epidemiological data and claimed that the data on HIV in AIDS patients contradicted the HIV-AIDS theory. In conclusion, Duesberg claimed that HIV played no functional role in AIDS and proposed that "the disease would then be caused by an as yet unidentified agent which may not even be a virus . . ." [17, p. 1215].

Instead of responding (in the journals or at meetings) to Duesberg's criticisms, AIDS researchers chose to ignore him. In justification, they claimed that his arguments were too erroneous to warrant a response. Furthermore, they argued that any response would publicize Duesberg's views and expose the public to potentially harmful ideas.

However, their strategy backfired. Although the scientific community shunned Duesberg, other communities provided a ready forum. Several gay newspapers, such as *The New York Native* and *Christopher Street*, applauded his efforts. The English television program 'Dispatches' also covered the story in a prize-winning expose entitled 'AIDS: the Unheard Voices.' Meanwhile, Duesberg regularly made public appearances to espouse his views. He spoke on radio

talk shows, appeared in CNN, and was lauded in an interview in *Spin*, a magazine that covers popular music and culture. At these appearances, Duesberg often used inflammatory language to attack the scientific community. At one point, Duesberg even offered to inject himself with HIV and jokingly remarked that "as long as it's not from Gallo's lab, I'll take it" [18].

Finally realizing that their silence was being interpreted as uncertainty, AIDS researchers organized to respond to Duesberg. The President's Commission on the HIV Epidemic invited Duesberg to testify in February of 1988, where the commission strongly criticized his viewpoints [19]. In April 1988, the American Foundation for AIDS Research sponsored a forum of virologists (including Duesberg), epidemiologists and pathologists to definitely resolve the debate. Each scientist countered Duesberg's contentions within their own field of expertise. At its conclusion, moderator Harold Ginsberg of the College of Physicians and Surgeons of Columbia University claimed that "[t]he large body of evidence presented appeared to resolve the Duesberg paradoxes, and considerable data were presented that fit HIV as the causative agent of AIDS according to the Henle–Koch postulates" [20].

Following their initial salvo, AIDS researchers stepped up their criticisms of Duesberg's arguments. Blattner, Gallo and Temin attempted to integrate the data from all the disciplines involved and presented a comprehensive response to Duesberg in a special policy forum section in *Science*. While they utilized data from many sources, Blattner and his colleagues cited the epidemiological data as the most convincing evidence of HIV's causative role in AIDS. In conclusion, they acknowledged that "many questions remain about HIV and AIDS," but staunchly declared that "a huge and continuously growing body of scientific evidence shows that HIV causes AIDS" [21].

The debate between Duesberg and Blattner *et al.* marked the beginning of a steady stream of scientific critiques of Duesberg's contentions. Among the laboratory researchers, Nobel laureate David Baltimore, a prominent retrovirologist addressed the controversy in an editorial to *The New England Journal of Medicine* [22]. Several clinical researchers also questioned Duesberg's arguments [23]. Chagrined at Duesberg's interpretation of their data and principles, epidemiologists entered the fray as well [24–26].

While they generally focused on the scientific merits of Duesberg's position, AIDS researchers also responded to Duesberg's sensationalized comments with their own rhetoric. Many accused Duesberg of endangering public health [27]. Several scientists also questioned Duesberg's qualifications to judge and interpret the data. George Klein, a virologist at the Karolinska Institute in Sweden, captured the sentiment when he described Duesberg as "a typical example of what the Swedish philosopher, Ingemar Hedenius, called 'charlatan camoufle,' the disguised charlatan, who is an expert in one field and uses his authority to make pronouncements about another that he does not understand" [28]. In particular, the AIDS researchers criticized Duesberg's especially stringent standards for establishing causation, and described his arguments as "quaint" [29] and "semantic and philosophical" [24, p. 112].

In light of the flurry of opposition from various scientists in different disciplines, Duesberg decided to comprehensively present his position in a review article to the *Proceedings of the National Academy of Sciences*. After an unusually extensive reviewing process and numerous exchanges with the editors, his article, entitled 'Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome: Correlation but not Causation,' was published in the February 1989 issue [30]. Rather than limit its arguments to Duesberg's field of expertise, retrovirology, the *PNAS* paper attempted to synthesize data from many different scientific disciplines, including virology, pathology, immunology, biochemistry and epidemiology. In a pattern typical of his subsequent papers, Duesberg systematically attacked each piece of evidence supporting the theory, while presenting his own counter-argument.

For the paper's centerpiece, Duesberg borrowed an epidemiological principle, the Henle–Koch postulates. Originally created by Jakob Henle, a nineteenth century physician, and later modified by Henle's student Robert Koch, the discoverer of the tuberculosis bacillus, the postulates consisted of three independent criteria to be satisfied before an agent could be causally related to a disease. Duesberg argued that HIV failed to satisfy any of the postulates; therefore, he contended, the virus could not be the causal agent of AIDS.

At the end of his article, Duesberg tentatively proposed an alternative theory of AIDS causation. Citing epidemiological data which indicated a 95% correlation between AIDS and certain risk factors, he proposed that AIDS may be caused by a new combination of these pathogenic factors. Without much data or support, the paper tentatively suggested that "[t]he habitual administration of factor VIII or blood transfusion or of drugs, chronic promiscuous male homosexual activity that is associated with drugs, numerous acute parasitic infections, and chronic malnutrition . . . appear to provide biochemically more tangible and plausible bases for AIDS than an idle retrovirus" [30, p. 761].

Although the overall response of the scientific community to Duesberg's theories was overwhelmingly negative, some supporters surfaced. These scientists did not necessarily support or agree with all of Duesberg's arguments, but applauded his efforts to force AIDS researchers to rethink and reassess their ideas. Some began to challenge the HIV-AIDS theory as well and offered their own theories of AIDS

causation. Most notably, Robert Root-Bernstein, a professor of physiology at Michigan State University and a recipient of a MacArthur grant, questioned the role of HIV in AIDS and offered a "multifactorial, synergistic theory of AIDS aetiology" [31]. Nevertheless, most scientists remained unconvinced by these arguments. However, their lack of support did little to dampen Duesberg's convictions, and Duesberg continued to present his views to any who would listen. In 1989, he heavily contributed to a book, entitled *AIDS: The HIV Myth*, by Jad Adams, a medical journalist [32]. In *Research in Immunology*, Duesberg presented his new, refined theory of AIDS causation, which proposed that AIDS was caused primarily by non-infectious agents such as drugs, overuse of antibiotics, AZT, malnutrition and parasitic infections [33].

At approximately the same time, Luc Montagnier, the original discoverer of HIV, published a paper in *Research in Virology*, which suggested that HIV may have a cofactor [34]. Building on the research of Shih-Ching Lo [35], head of the AIDS Pathology Division of the Armed Forces Institute of Pathology who had identified a unique mycoplasma among several AIDS patients, Montagnier openly questioned the sufficiency of HIV in causing AIDS.

Although Montagnier and Lo maintained that HIV was a necessary agent for AIDS, their suggestions created a furor among the AIDS scientific community over the precise role of HIV in AIDS. Seizing on this uncertainty, Duesberg in conjunction with Bryan Ellison a doctoral student in molecular and cell biology, published a comprehensive description of his theory in the *Policy Review*, a politically conservative journal sponsored by the Heritage Foundation [36]. The paper not only expanded its critique of the HIV-AIDS theory, but discounted Montagnier's cofactor theory as "an invention to try to fill the gaps in any theory that blames the AIDS disease on a microbe." Alternatively, they claimed that their "risk hypothesis explains the many paradoxes of AIDS and HIV" [36, pp. 49–50].

The *Policy Review* article provoked a flurry of responses and "received more comments, both positive and negative, than on any other article in recent memory" [37]. Not surprisingly, virologists, epidemiologists and clinicians all wrote to criticize Duesberg's arguments. These groups were joined by various citizen groups, such as the American Council on Science and Health, in their denunciations of Duesberg and his views. These detractors were countered by a number of supporters who either questioned the HIV-AIDS theory or applauded Duesberg's efforts to challenge it.

Duesberg later offered more detailed arguments for his theory in *Biomedicine and Pharmacotherapy* [38]. In the article, he provided a lengthy list of data and studies which he claimed supported his drug-AIDS hypothesis. One of his latest papers, entitled 'Latent Viruses and Mutated Oncogenes: No Evidence for Pathogenicity' [39] matches the HIV-AIDS theory against his drug-AIDS hypothesis and claims the superiority of the drug-AIDS theory.

While Duesberg's theories of AIDS causation have not been accepted, he continues to receive much attention from the popular press and the AIDS research community [40]. More recently, other scientists have begun to openly question the previously accepted theory. A group of 50 scientists, including Kary Mullis, the inventor of the polymerase chain reaction (PCR), have joined to form an international group, called The Group for the Scientific Reappraisal of the HIV/AIDS Hypothesis. Some of these scientists appeared along with Duesberg and his critics in a recent national television news program on HIV and AIDS in March 1993.

Despite the growing skepticism, most researchers remain convinced that HIV plays a significant role in AIDS and still regard Duesberg's actions as a danger to society. These sentiments toward Duesberg are clearly reflected in the response to an editorial written by John Maddox, the editor of *Nature*. In the editorial, Maddox concluded by suggesting that "Duesberg will be saying, I told you so" [41]. His comments sparked an outrage among scientists who called the article "outrageous, fatuous, woolly and confused" [42]. While Maddox's recognition of Duesberg suggests that scientists are now paying attention to Duesberg's arguments, Maddox's eventual retraction indicates that most scientists are hardly ready to abandon the HIV-AIDS theory.

STYLES OF SCIENTIFIC PRACTICE

Duesberg's constant attacks on the accepted theory of HIV-as-cause forced HIV-AIDS researchers to respond by making their arguments and assumptions explicit. Indeed, Duesberg's efforts and the resulting controversy illuminate the different styles of scientific practice through which scientists verify their conclusions. By examining the responses of HIV-AIDS thesis supporters to Duesberg's critiques, we have constructed an 'epidemiological' style of practice that frames their arguments. However, first we must specify what we mean by styles of practice.

Our styles of practice refer to the plurality of sciences and their attendant methods for verifying theories. In contrast to the focus of idealist philosophers on theory production and validation as forms of logic or ways of thinking, our styles of practice also include the activities of hands and eyes and the discourses between multiple actors in diverse situations. Styles of practice are historically located and collectively produced work processes, methods, and rules for verifying theory. As such, they are unattached to any notion of objective metaphysical truth or to the notion of a single 'scientific method.' Style of practice implies that practices of theory construction, adjudication, and maintenance are situated actions, that they can and do change, and that

they change in interaction with the many agents involved in their fabrication. The study of practices also widens our focus from a narrow view of 'thinkers' and 'reasoners' to the many other participants in science-making.

Our styles of scientific practice have much in common with philosopher of science Ian Hacking's "styles of scientific reasoning." For Hacking, a style of scientific reasoning is a way of thinking, talking, arguing, showing, and, I would add, crafting [43]. Hacking discusses several styles that have developed through time to become substantially different from each other in several ways, each with its own unique history. Each style became associated with a unique set of techniques, laws, and objects that was used to authenticate or verify its theories and facts. According to Hacking, each style of reasoning is historically based but can and has become autonomous of its historical origins to stand alone as a canon of objectivity.

> Every style comes into being by little microsocial interactions and negotiations. It is a contingent matter . . . Each style has become *what we think of* as a rather timeless canon of objectivity, a standard or model of what it is to be reasonable about this or that type of subject matter. We do not check to see whether mathematical proof or laboratory investigation or statistical 'studies' are the right way to reason: they have become (after fierce struggles) what it is to reason rightly, to be reasonable in this or that domain [7, p. 10, emphasis added].

When using Hacking's framework, we assume no one-to-one mapping of group membership to a particular world of practice and arguments within styles of practice. Although styles of inquiry have their bases in socio-historical times, particular styles of practice continue on through time and co-exist with other styles of practice in scientists' adjudicatory actions. This is evident in the actions of Duesberg who appeals to different styles of practice when constructing his arguments and criticisms. We see in his example that there is no one-to-one correspondence between type of scientific training (or membership in a particular discipline or line of research) and a particular style of scientific practice [44]. Researchers can argue within the frame of a particular style of practice even if others from the same field do not. Scientific practice is very heterogeneous. For example, both Gallo and Duesberg are retrovirologists, yet they take opposing positions in this controversy. Indeed, most retrovirologists argue that the overwhelming evidence supporting their view that HIV causes AIDS comes from epidemiological evidence.

We borrow from Hacking's notion of styles of scientific reasoning its way of handling the problem of realism and objectivity in terms of practices rather than in terms of logical rules of argument. Hacking argues that each style of reasoning has its own characteristic 'self-stabilizing' techniques, developed over a long time, which are used to 'authenticate' theories within the frame of reference of the style. Thus scientific reasoning is a historical and collective set of practices unattached to ideas of objective metaphysical truth or a single "scientific method" [45]. However, they are necessary for understanding objectivity. "My styles of reasoning . . are part of what we need to understand what we call objectivity. This is not because styles are objective (i.e. we have found the best impartial ways to get at the truth), but because they have settled what it is to be objective (truths of certain sorts are just what we obtain by conducting certain sorts of investigations, answering to certain standards)" [7, p. 4].

Using this framework of styles of scientific reasoning, Hacking then defines verification as embedded in a particular style of scientific reasoning. A statement, theory, or fact is 'true to' a particular set of self-stabilizing techniques within a particular style of scientific reasoning [46].

> The truth of a sentence (of a kind introduced by a style of reasoning) is what we find out by reasoning using that style. Styles become standards of objectivity because they get at the truth. But a sentence of that kind is a candidate for truth or falsehood only in the context of the style. Thus styles are in a certain sense 'self-authenticating.' Sentences of the relevant kinds are candidates for truth or for falsehood only when a style of reasoning makes them so. . . There simply do not exist true-or-false sentences of a given kind for us to discover the truth of, outside of the context of the appropriate style of reasoning.
>
> The apparent circularity in the self-authenticating styles is to be welcomed. It helps explain why, although styles may evolve or be abandoned, they are curiously immune to anything akin to refutation. There is no higher standard to which they directly answer. The remarkable thing about styles is that they are stable, enduring, accumulating over the long haul. Moreover, in a shorter time frame, the knowledge that we acquire using them is moderately stable. It is our knowledges that are subject to revolution, to mutation, and to several kinds of oblivion; *it is the content of what we find out, not how we find out that is refuted* [7, p. 13; emphasis added].

We next use the framework of styles of practice to examine the HIV-AIDS controversy.

STYLES OF SCIENTIFIC PRACTICE IN THE HIV-AIDS CONTROVERSY

As we have seen in our story of the debate, Peter Duesberg scoured the published articles for data. He analyzed each set of data and argued against the claims of AIDS researchers that the data supported their conclusion that HIV causes AIDS. Duesberg also pointed to what he perceived as conflicts between the different kinds of data, unavailable data, and inadequacies in experiments and studies producing the data. When researchers responded that even retrovirological data had to be understood in conjunction with epidemiological knowledge, he began to analyze epidemiological data and argue against that information. In response, AIDS researchers maintained that he did not know how to properly interpret epidemiological data. Duesberg in turn accused them

of interpreting their data to fit their theory. Will the real scientist please stand up?!

We propose that all of these debaters are real scientists. When applied to the current controversy about the etiology of AIDS, the framework of styles of practice allows us to view different sides of the controversy as speaking in different languages of verification. That is, they use and invoke different styles for adjudicating 'truth.' We are concerned here with different rhetorics and rules for judging the adequacy of statements that are embedded in different styles of practice. (This controversy would be a deadly game if it were merely rhetorical jousting.) In the early years of the debate, Duesberg framed his criticisms of the HIV-AIDS thesis within a *laboratory style of practice*. In contrast, most HIV-AIDS researchers employed and still employ what we call an *'epidemiological' style of practice*. By appealing to different styles of practice, the two sides of the controversy came to entirely different conclusions when evaluating the same data. This is in part why one side's arguments are often orthogonal to the other's. Despite the acrimony, both sides are sincere about their respective conclusions and positions. We first briefly examine the laboratory style of practice and then move on to discuss the epidemiological style of practice in more detail.

Laboratory style of practice

In the early part of the controversy, Duesberg's criticisms of the HIV theory were framed in terms of a type of verification we call a laboratory style of practice. In his evaluation of published data on HIV as the cause of AIDS, he pointed to inaccurate or incomplete data and to contradictions between data. Duesberg refused to allow accumulation of imperfect data as acceptable verification of the theory. His critique appears to be 'reasonable' when viewed within Hacking's formulation of a laboratory style of reasoning "characterized by the building of apparatus in order to produce phenomena to which hypothetical modeling may be true or false, but using another layer of modeling, namely models of how the apparatus and instruments themselves work" [7, p. 6]. New phenomena and models are meshed together through such apparatuses. If such apparatuses cannot be constructed or do not perform adequately, then the argument, model, or theory is not 'vindicated.'

Hacking argues that theories are 'self-vindicated' through the meshing of data, instruments, and theory in laboratory sciences [47]. He proposes that the relationship between observation and theory in mature laboratory sciences is mediated by a number of elements and activities whose particular ways of cohering are not preordained in any particular way. Instead, theory and observation are connected to each other through processes of *meshing* together these mediating elements including ideas (questions, background knowledge, systematic theory, topical hypotheses, modeling of the apparatus), things (target, source of modification, detectors, tools, data generators), and marks and the manipulation of marks (data, data assessment, data reduction, data analysis, and data interpretation). These elements and their 'mesh' with each other are constructed through time to 'fit' each other. In other words, they are *co-produced* [48].

A theory, then, is vindicated by or 'true to' its set of ideas, activities, markers, and marks. Hacking argues that "our preserved theories and the world fit together so snugly less because we have found out how the world is than because we have tailored each to the other" [49].

> Theories are not checked by comparison with a passive world with which we hope they correspond. We do not formulate conjectures and then just look to see if they are true. We invent devices that produce data and isolate or create phenomena, and a network of different levels of theory is true to those phenomena. Conversely we may in the end count them as phenomena only when the data can be interpreted by theory. Thus there evolves a curious tailor-made fit between our ideas, our apparatus, and our observations. A coherence theory of truth? No, a coherence theory of thought, action, materials and marks [49, pp. 57–58].

Hacking specifically refers to the meshing of practices, beliefs, and tools with each other through the developmental history of a mature laboratory science. He [49, p. 61] acknowledges in his text that he confines his discussion (except for a few qualifications) to the 'internal' workings of a laboratory science. His goal is to attempt to explain the stability of theory within a particular laboratory science, and he refrains from arguing that a stable theory extends outside that science to explain the 'real world' in any metaphysical sense. His is a self consistent system where theory is 'vindicated' within this internalist system [50].

Indeed, a laboratory science like molecular biology is amazingly internally consistent. Molecular biologists have achieved a tightness of fit between ideas, tools, and data that is rare in the history of biology. Duesberg, practicing in contemporary laboratory sciences of molecular biology and virology, called for a similar tightness of fit of AIDS research in the early stages of the debate. However, internally consistent theories are no more, and no less, 'true' than theories produced in less internally consistent systems [7, 24]. Epidemiology, in contrast, has a tradition of another style of practice and theory adjudication that has been constructed over time to adapt to the contingencies associated with the study of diseases across populations. If populations of people were like experimental 'nude' mice in environmentally controlled rooms, epidemiology could perhaps achieve the internal consistency of laboratory sciences [51]. However, such artifice would defeat the purposes of epidemiologists involved in controlling and preventing the further spread of diseases. We now turn our attention to the practices of epidemiology.

ELEMENTS OF AN EPIDEMIOLOGICAL STYLE OF PRACTICE

While both sides of the controversy speak in the language of theory adjudication, they use different rules for adjudicating 'truth.' When challenging HIV-AIDS researchers to prove their theory, Duesberg asks them to satisfy rules of verification that they argue have never been applied in the strict sense required by Duesberg.

Instead, scientists working on problems like AIDS construct a mosaic framework of data, materials, technologies, and knowledges produced by different expertises or methodologies. No one element or piece of information can adequately define the problem or the etiology. Only the composite view presents the viewer with a discernible picture or pattern. This mosaic aspect is one element of what we call the epidemiological style of practice. The mosaic includes retrovirological experiments, statistical and epidemiological collection and analyses of CDC data, sociological and anthropological studies of groups and cultural practices, and medical case studies. Unlike the verification exercise of triangulating data to confirm a single point, researchers are actively combining ideas, data, and apparatuses in ways not circumscribed by any one discipline. They move across disciplines and specialties in constructing their arguments for the 'truth' of a particular theory. They compile, juxtapose, and connect data in ways not governed by any single discipline and then handle the incongruities and gaps through interpretations based on tacit knowledges. Perhaps the best discipline to represent this style of practice is epidemiology, hence our choice of nomenclature.

In the case of AIDS (and perhaps in the study of most diseases and other such practical problems), the production of knowledge spans many biomedical research disciplines (e.g. virology, cell biology, immunology), medical research and clinical practices, public health sciences (e.g. epidemiology, biostatistics), social sciences (sociology, psychology, anthropology, economics), legal-ethical worlds, as well as patient worlds and other affected populations (e.g. gays, hemophiliacs and other recipients of blood products, intravenous drug users, and their self-appointed or elected spokespeople, government agencies (e.g. Center for Disease Control), and private industry (e.g. health insurance companies, blood banks). One project, the San Francisco Men's Health Study, alone employs, among others, epidemiologists, physicians, two virologists, an immunologist, a pathologist, a sociologist, a psychologist, an anthropologist, and a dentist. With theories constructed and verified between disciplines, the linking and meshing of data with theory becomes more complex to achieve and to study than in laboratory sciences like molecular biology. Adjudicating between and attempting to mesh together their different kinds of data, different forms of argument, different units and levels of analysis, different temporal orientations, and different orientations toward anomalies require, among other things, time, data processing and interpreting efforts, research funds, and many situations of collective work [52].

We are interested in how the links between observation and theory are constructed, not within a single mature laboratory science, but between clinical practices, basis research, and epidemiological research in these complex situations; between field and case study information and statistics; between historical understanding and contemporary problems. The very organization and nature of biomedical science, and especially epidemiology, indicates inter-world activities and joint work. These situations therefore are good places/spaces for sociologists of science to study the negotiations between and meshing of practices and knowledges produced by different *worlds of practice* [53].

Constructing the disease entity, tracking its course, and searching for one or more causal agents demanded interaction between many different worlds of practice [54]. The very construction of AIDS as a single problem was a collectively produced phenomenon. In considering this complex set of interactions, one can argue that a stable theory was established relatively quickly (although most observers argue that locating a 'cause' took longer than it should have). Duesberg argues that the HIV thesis was presented by political fiat, but political fiat alone does not explain why most participants in AIDS research in the past nine years came to support the thesis that HIV causes AIDS. AIDS researchers contend that even though they have not been able to demonstrate exactly *how* HIV causes AIDS, this does not mean that HIV does *not* cause AIDS. We argue that this acceptance is based on a process of self vindication, although the meshing processes are more complex than in the case of laboratory sciences because different worlds are attempting to mesh their different sets of practices and products. HIV-AIDS researchers have constructed a self-consistent system wherein HIV is a necessary cause of AIDS from a mosaic or pathwork quilt of evidences from different expertises, different scientific and social scientific disciplines, and medical practices.

Defining an epidemiological style of practice

We use various discussions of 'what is epidemiology' from several journals of epidemiology, medicine, and public health, histories of AIDS epidemiology, Duesberg's arguments and responses to his arguments, and interviews with practicing HIV-AIDS researchers to begin to define some elements of our 'epidemiological' style of practice. We do not intend to write a history of epidemiology here. Our purpose is to use these discussions to define a style of practice within which HIV-AIDS researchers characterize and defend their view that HIV causes AIDS. We use the histories of epidemiology to understand and display

the rules of verification constructed and used by HIV-AIDS researchers to argue that HIV causes AIDS in the current debate.

We argue that epidemiologists and other AIDS researchers have responded to the disease in a style of practice that has developed (in process and content) during the last century. AIDS epidemiologists understand that a particular epidemiological style was put into practice in the effort to deal with the unknown novel disease and its subsequent classification as a chronic disease [55]. We would also add that the history of epidemiology is one of recurrent major and minor changes [56]. Indeed, discussions and debates about what epidemiology is have appeared in biomedical journals with amazing regularity. Prominent discussions occurred in the 1930s, 1940, 1950s, 1960s, and the 1970s [57–62].

Some epidemiologists promote the idea that epidemiology is a scientific method of investigating etiology in the biomedical sciences (and less an autonomous discipline) [63]. Morris argues that: "Epidemiology [is] a procedure for finding things out, of asking questions, and of getting answers that raise further questions—that is, as a *method* [as well as] the *results*, the information, obtained in reply" [61, p. 396]. One thing that epidemiology attempts to answer is the question of disease etiology. As Morris points out,

> [t]he main function of epidemiology is to discover groups in the population with high rates of disease, and with low, so that causes of disease and of freedom from disease can be postulated... The biggest promise of this method lies in relating disease to the ways of living of different groups, and by doing so to unravel 'causes' of disease about which it is possible to do something. The great advantage of this kind of approach to prevention is that it may be applicable in the early stages of our knowledge of diseases, to disrupt the pattern of causation before the intimate nature of disease is understood. Sufficient facts may be established for this by epidemiological methods alone, or in combination with others. The opportunity may thus offer to deal with one 'cause,' or with various combinations of causes [61, p. 399].

However, according to (my translation of) historian Fagot-Largeault, this method is not without discouragement. "It is far from the judicious order of a deductive discipline. The simple collection of facts, and the treatments given, hide formidable problems" [56, p. 166]. Some of these formidable problems are documented in the history of AIDS epidemiology, which we discuss below.

We propose that epidemiology follows a particular style of practice that can be characterized as a mosaic constructing industry, where incongruities are common and do not stop the flow of action. Consider, for example, the document on "HIV Infection and Its Epidemiology" published by the National Academy of Sciences [55]. Here are some key phrases of the NAS committee's decision, after evaluating available data, that "the evidence that HIV causes AIDS is scientifically conclusive".

> That a particular organism causes a disease is demonstrated by a confluence of evidence linking the two. HIV and AIDS have been linked in time, place, and population group...
>
> The conjunction heralded by the joint appearance of HIV and AIDS has been confirmed by their continued association. HIV seroposivity rates in defined subpopulations of homosexual men in San Francisco and New York City and in IV drug abusers in New York City are associated with later cases of AIDS in the same groups...
>
> The virus has been isolated from persons with AIDS; as assay techniques have improved, close to 100% of affected individuals can be found to harbor the virus... The virus is not found in persons who are not at risk for infection [64].
>
> Perhaps the clearest evidence linking HIV to AIDS is to be found in the tragic results of blood transfusions in the United States and around the world. The transmission of HIV in contaminated blood and blood products has been clearly linked to AIDS...
>
> The causal role of HIV in AIDS is also supported by the high risk (30–50%) of perinatal HIV transmission from an infected mother to her infant... and the subsequent diagnosis of AIDS in the infected infants.
>
> The pathogenesis of HIV infection—how the organism causes disease—is still incompletely understood... A complete understanding of a disease's pathogenesis, however, is not a prerequisite to knowing its etiology [55, pp. 74–76].

We point to several key elements of the argument that HIV causes AIDS. These include: quantitative population analyses associating HIV with AIDS at particular times, places, and within particular population groups; the continued association of these variables over long periods of time; the clinical production of information on individual symptoms, progression, and pathogenesis; clinical diagnoses to produce AIDS 'cases'; laboratory technologies, especially molecular and biochemical assays for detecting the presence or absence of HIV in individuals; laboratory research on pathogenesis *in vitro*; and combinations of the above to study transmission patterns and rates of transmission of HIV. According to the NAS committee, this combination of findings satisfactorily confirms the theory that HIV causes AIDS.

We add another element that has played a critical role in AIDS research: political, medical, and public health efforts to stop transmission and to treat infected persons. Epidemiology is a scientific practice tied to practical action. Prevention and control are two applied arms of this discipline. Epidemiologists are currently preparing protocols for vaccination trials (even before potential vaccines are available because of the inordinate amount of work it takes to coordinate such trials) [Winkelstein interview 1992]. They are working jointly with public policy groups and public health departments to track risk populations and to educate all populations [65]. Epidemiologists also continue to work with clinicians to study the progression of the disease in patients. That is, besides working in conjunction with laboratory

sciences like retrovirology and immunology, epidemiology has an applied arm that requires interaction with many different groups and practices far from the laboratory. In the history of epidemiology, this interaction is posed perhaps more positively with post-war enthusiasm by Morris.

> Epidemiology . . . as a tried instrument of research—with its modern developments in sampling and surveys, small-number statistics, the follow-up of cohorts, international comparisons, field experiment, and family study; and with its extensions to problems of genetics as well as environment, to physiological norms as well as disease, the psychological as well as the physical, morbidity as well as mortality—*epidemiology now offers* the possibility of a new era of collaboration between public health workers and clinical medicine. Such a collaboration could be on equal terms, *each making their particular contribution to the joint solving of problems*. . . . Epidemiology, moreover, is rich with suggestions for clinical and laboratory study, and it offers many possibilities for testing hypotheses emerging from these [61, p. 399; emphasis added].

These interactions with other practices also incorporate methodological problems. For example, epidemiologists are linking data expressed in non-equivalent terms produced by different practices such as case studies, quantitative analyses, and laboratory experiments. One can ask sociological and philosophical questions about how epidemiologists construct link between such non-equivalent forms of data. How do these researchers choose what counts as acceptable evidence? How do they select and mesh together different forms of argument, different units and levels of analysis? How do they decide which technologies to use, which not to use? How do they decide what is disease, as new molecular technologies allow them to define novel 'diseases?'

Invoking and constructing histories of epidemiology as evidence

We argue then that the conclusion that HIV causes AIDS is a product of multiple and interactive practices constructed through the history of the study of infectious and chronic diseases. To focus our discussion of how these interstitial (inter-world) incongruities have been handled in the construction and verification of disease causation, we will analyze the different uses and interpretations of Koch's postulates made by epidemiologists and by Duesberg. One of Duesberg's major arguments is that laboratory research in retrovirology has been unable to specify the means by which HIV causes AIDS [66]. While retrovirologists have demonstrated pathogenesis in cell cultures, Duesberg did not accept it as adequate verification that HIV causes AIDS because an intact immune system is nonexistent in laboratory models [67]. The ability to reproduce AIDS (as exhibited in humans) in an animal model would be an acceptable verification. In making this argument, Duesberg invokes Koch's postulates for relating causative agents to disease.

An example: historical revisions of Koch's postulates. We present the Henle–Koch postulates as our example of an epidemiological principle under construction and contestation. During the late nineteenth century, Jakob Henle, a physician in Zurich, proposed some principles for analyzing the role of 'Microscopic forms' in disease causation. Based on these principles, Henle's student Robert Koch, proposed three independent criteria to be satified before an agent could be causally related to a disease. The three postulates, known as the Henle–Koch or more commonly as the Koch postulates, became "our classical point of reference in relating causative agents to disease" [57, p. 250]. While AIDS researchers used the postulates to support HIV as the causal agent of AIDS, Duesberg argued exactly the opposite case.

Koch's postulates state that to prove that an infectious agent is the cause of an illness, it is necessary to establish that: (1) The parasite is present in every case of the disease under appropriate circumstances. (2) The parasite should occur in no other disease as a fortuitious and non-pathogenic parasite. (3) The agent must be isolated from those infected subjects, cultivated *in vitro* (in pure culture), and induce the disease when introduced into healthy subjects.

According to Duesberg, the scientific data demonstrated that HIV did not satisfy this postulate. First, he argued that there was little evidence of HIV in most patients with AIDS and claimed that only 40% of the patients in San Francisco and 7% of the patients in New York had been confirmed to be HIV positive [30, p. 756]. Most of Duesberg's criticisms were related to the last postulate. Koch's third criterion called for the induction of the disease in a suitable host upon infection with the hypothesized agent. Duesberg argued that the infection of chimpanzees with the virus did not cause AIDS-like symptoms, despite the successful induction of HIV antibodies. Second, he claimed that the evidence of accidental infections of HIV in health-care workers was inconclusive, because most of these workers had not developed AIDS and the purported examples remained unconfirmed. Finally, he contested the evidence of transfusion-related AIDS cases. Citing the possible presence of other toxins in screened blood (due to inadequate screening techniques) and the lack of controlled studies, Duesberg rejected the data on blood transfusions as "presumptive" [30, p. 757].

In response, HIV-AIDS researchers argued that Duesberg was asking for an unrealistic application of the Henle–Koch postulates. They contend that histories of epidemiology and disease causation demonstrate that Koch's postulates are historical objects that have been modified, interpreted, and used differently in different historical periods (and perhaps also by different practitioners in diverse geographical locations). The following is a brief acount of changes in the use and interpretation of Koch's postulates in

the 1900s. It is an example of changes over time in the rules of verification in epidemiology.

Even Koch himself recognized that his postulates were fulfilled by a few 'classical' bacteria (e.g. those causing anthrax, tuberculosis, erysipelas, tetanus, and many animal diseases), but not by others (e.g. those causing typhoid fever, diphtheria, leprosy, relapsing fever, and Asiatic cholera) [24, 57]. In addition, Koch did not understand that healthy persons could carry bacteria, a fact that would create situations where his third postulate would not apply [68]. This perhaps explains why, according to Evans, "Koch emphasized that not all the criteria were necessary for proof and that just the first two were sufficient" [57, p. 250].

With the discovery of diseases associated with viruses in the 1900s, Koch's postulates were re-negotiated. Thomas Rivers introduced revisions to the original Koch's postulates, including: (a) More than one agent might be needed to produce a given disease; (b) Asymptomatic carriers existed; and (c) Antibody to the disease should appear during the course of the illness, thus adding a dimension of immunological proof. Rivers' revised "Koch's postulates" also included a statistical dimension: (1) The viral agent is associated with the illness with a certain statistical regularity; (2) One can isolate the agent, cultivate it under appropriate circumstances, and reproduce the illness with a certain regularity (in controlled trials); and (3) Even if the illness does not occur experimentally, the appearance of antibodies gives evidence for viral activity.

In the 1950s, the introduction of electron microscopy inspired another renegotiation of Koch's postulates by providing evidence that many viruses were sometimes found to co-occur in sick as well as in healthy people. In 1957 Robert Huebner "indicated that the presence of many viruses—even in normal people—made the identification of the presence of virus of low order in establishing causation and one could derive spurious causative associations if you based the conclusions on this fact alone. He recognized that some infections were due to multiple viruses, and that sometimes viruses could produce chronic disease, or carrier states or that viral reactivation occurred" [59, p. 251]. In writing his 'bill of rights' for prevalent viruses, Huebner reorganized Rivers' postulates. To provide evidence that the virus was responsible for the infectious syndrome, "it was necessary to establish the reality of the viral agent and describe its characteristics and to possess, if not the proof that it induces the syndrome, at least immunological proof of its action. But the better argument in favor of causality was the production of a specific vaccine capable of protecting people against the disease (proven in controlled tests). [Huebner's 'bill'] emphasized that the mere presence of the virus should not be regarded solely as the basis for etiology and he introduced epidemiologic principles by longitudinal and cross-sectional studies as an element of proof in causation . . . Huebner also emphasized that one needed money in order to accomplish the establishment of proof and included this as a ninth criterion for etiologic studies" [59, p. 251].

Over the next 10 years, researchers' understandings of the activities and effects of infectious agents changed [57]. They came to believe that: the same clinical syndrome could be produced by a variety of different agents (bacteria, virus, or parasites, either working together or separately); the same agent could cause different syndromes in different locations; different agents predominated different clinical cases depending on epidemiological circumstances (age, season, population groups); the host response to a given virus would vary from one setting to another; finally, "the agent alone was a necessary but not sufficient factor needed to cause most diseases. Cofactors and the susceptibility of the host were of key importance in the occurrence of *clinical* illness" [59, p. 251].

In addition, in a number of cases such as Burkitt's lymphoma which is thought be caused by the Epstein–Barr virus, the causal agent could not be found either through clinical analysis or via sophisticated laboratory technologies. The cases were later followed by the establishment of an immunologic proof of causation (which is specifically not accepted by Duesberg), where the discrete presence and absence of antibodies at different times were considered to indicate the causal agent. Evans uses the example of the Epstein–Barr virus to argue that Koch's postules and other such criteria of proof should change with changing technologies.

> It is a particular irony that Dr [Werner Henle], who is the grandson of Jakob Henle, established the [immunological proof of the] causative relationship of EBV to infectious mononucleosis without fulfilling a single one of the postulates set up by his grandfather and by Robert Koch. *This serves to emphasize that we must change our criteria with our technology* [59, pp. 252–253; emphasis added].

The 1970s presented a new challenge to the application of Koch's postulates in disease causation in the form of kuru, a disease defined in New Guinea, and Creutzfeld–Jakob disease, a pre-senile dementia. D. Carleton Gajdusek proposed that they were caused by lentiviruses, or slow viruses, that have long incubation periods before the disease symptoms actually appear [69]. These diseases presented particular difficulties to verification of causation "because of their long incubation periods, their relation to chronic neurologic disease, and most importantly from the standpoint of the Henle-Koch postulates, the fact that the agents could not be isolated in tissue culture in the laboratory" [59, p. 252]. Furthermore, these unique agents did not produce an immune response and were highly resistant to a great variety of physical and chemical agents. In 1974 Richard Johnson and Clarence Gibbs proposed a new set of criteria for establishing causation to deal with these novel viruses. The guidelines were: (1) In at least two independent laboratories, the agent must either be

consistently shown to be transmitted in animals, or the virus must be identified in cell cultures using techniques for showing high specificity. (2) The agent should be serially transmissible in experimental animals with fitered material. (3) The virus is not found everywhere, that is, that similar results should not be obtained from normal tissues. Unfortunately, even if research could be carried out, these criteria are very difficult to fulfil [56, 58, 59].

In the 1960s and 1970s, research on certain viruses as possible causes of cancer produced similar difficulties and led to calls for a new set of criteria for verifying causation. These viruses, called retroviruses, also apparently had long incubation periods before the onset of the disease. "But proof of causation was difficult because they are common and ubiquitous viruses, probably require co-factors, and there are difficulties in reproduction of the cancer in animals. In addition, human volunteer studies are not possible. There is also the probability that the cancer may have different causes in different geographic areas or under different epidemiologic settings" [59, p. 252].

In 1976 and 1978 Evans summarized and unified the changes introduced in the previous guidelines for attributing causation. He called his new guidelines "a unified concept of disease causation," in part because he included causation of chronic diseases in his unified concept [70]. The main features of these guidelines for attributing disease causation were:

> (1) The prevalence of the disease should be higher in those exposed than in those not exposed, (2) that exposure to the putative cause should be present more commonly in those with the disease than in those without the disease, (3) that the incidence should be higher in persons who are so exposed than in those not exposed as shown in prospective studies, (4) that exposure to the suspected factor should precede the disease, (5) that there should be a measurable biologic spectrum of host responses, (6) that experimental reproduction of the disease should be demonstrated, (7) that elimination of the putative cause should decrease the incidence of the disease, and (8) that prevention or modification of the host response should decrease or eliminate the expression of the disease [59, p. 254].

Evans added a qualifier to his guidelines: "just as the Henle–Koch postulates cannot be regarded with any finality, so too, these concepts should be taken only as guidelines, subject to our changing knowledge of technology and causation" [59, p. 254]. He went on to argue that the original Henle-Koch postulates had many limitations, and he instead prescribed a *multifactorial approach to disease causation.*

> Fulfillment of the postulates is certainly reasonable grounds for accepting a causal role of the putative agent but lack of fulfillment of the postulate should not exclude such a relationship... Most infectious agents are a necessary but not sufficient cause of disease; indeed many viral infections are inapparent. Causation in both infectious and non-infectious disease involves a complex interplay of agents, environmental, and host factors. The latter include the host's immunologic status, genetic background, socioeconomic level, hygienic practices, behavioral patterns, age at the time of exposure and the presence of co-existing disease. Different qualitative and quantitative mixes of the agent, environment, and host may result in the same clinical pathological diseases under different circumstances [59, p. 254].

Evans' 1978 article ends with a list of possible sources of and paths via which new infectious diseases could develop. He warned readers that "we may be faced in the future with a variety of new and unrecognized infectious disease whose etiology must be established. Proof of this relationship must be based on common sense, good guidelines of causation appropriate to existing technology and a keen sense of the biologic basis of disease" [59, p. 256]. Little did he know that just 5 years after this statement's original publication in 1976, patients with 'AIDS' would begin to appear in doctors' offices in the United States.

(Re)writing histories of epidemiological practice

The point of our review of this history of epidemiology is that, when Duesberg appeals to Koch's postulates as they were strictly written or even as Koch interpreted them in his time, he is appealing to an object, a set of rules, non-existent in current epidemiological practice. As interpreted and used by epidemiologists practicing in the 1980s and 1990s, Koch's postulates support the verification of HIV as the cause of AIDS. For example, epidemiologist Evans refutes Duesberg's use of the exact terms of Koch's original postulates by invoking the historical redefinitions of Koch's postulates in epidemiological practice. "Duesberg says that HIV does not fulfil the Henle–Koch postulates... It is important to review these classical criteria in historical perspective" [24, p. 107]. Evans goes on to recount the history of changes in the Henle–Koch postulates. He especially notes the specific biological, clinical, environmental and pharmacological historical 'discoveries' that researchers argue led to the periodic revisions of the postulates that relate to the current AIDS situations. Analyzing the evolution of the Henle–Koch postulates "from the standpoint of an epidemiologist" [24, p. 109] and considering changing technology and new knowledge, Evans reiterates his "unified concept for causation." Applying his framework to the HIV question, he recognized that the data on HIV could not absolutely satisfy all the criteria, but declared such a strict adherence to principle unnecessary when the "available evidence overwhelmingly supports the concept that HIV plays a critical and necessary role in the pathogenesis of AIDS" [24, p. 112]. Finally, Evans is quite clear that "any guidelines of causation [including his own] must change as new technology and new concepts of pathogenesis develop" [24, 109].

Thus, in response to Duesberg's criticisms of their conclusions, *epidemiologists invoked their history of epidemiology, including the history of changes in Koch's postulates. Duesberg then responded in turn to epidemiologists' invocation of history by constructing his own history of epidemiology.* Indeed, in a May 1992 colloquium presented to the University of Cali-

fornia, Berkeley, statistics department, Duesberg was explicit about his intent to deconstruct and reconstruct the history of epidemiology to demonstrate what he views as shortcomings in the arguments presented by HIV-AIDS researchers. "So how could science go so wrong? Since science has become in fact the only religion that all of us believe in, now we believe in the infallibility and unbiasedness of science and here is an example—here are examples that this did happen before. Even in this century there are numerous examples where science has gone wrong" [71].

Rather than argue within epidemiologists' history of epidemiological rules for verification, Duesberg has accumulated information to deconstruct their history of epidemiology and construct his own version. He refused to accept the epidemiologists' history not of only Koch's postulates, but also of infectious diseases. He has supplemented his experience with retroviruses with knowledge about the processes, activities, and consequences of viruses in disease causation gained from perusing the bookshelves of the Public Health library at the University of California, Berkeley. In a 1991 letter to *Science*, Duesberg presented a version of his history of disease research, focusing on examples of failures due to proceeding with the assumption that germs are the cause of diseases.

> Other examples demonstrate that the ever-popular germ theory has at times 'remained a candidate' far too long, until finally disproved at great cost to the affected people. In the United States tens of thousands died unnecessarily in the 1920s because pellagra was considered infectious by the U.S. Public Health Service, until Joseph Goldberger proved it to be a noninfectious vitamin B deficiency. Indeed, the disease was said to be transmitted by 'poor hygiene' among corn farmers in the South—the primary risk group for pellagra. In Japan, at least 10,000 suffered in the 1960s and 1970s from a drug-induced neuropathy, including blindness, that had been misdiagnosed as a viral disease for more than 10 years [72].

In the May 1992 statistics colloquium, Duesberg added more items to his list of epidemiological failures. For example, he argued that the slow (lenti-) virus which Gajdusek proposed as the cause of 'kuru' disease in New Guinea has never been found and that the disease has since disappeared. Duesberg contended that cannibalism, the suggested means of transmission of the disease, has since been considered to be a falsification. He further suggested that the disease was a genetically based neurological disorder and not an infectious disease. Using his biting wit, Duesberg stated that "the Nobel prize still exists, but everything else is not there." Another example on Duesberg's list was Burkitt's lymphoma, said to be caused by the Epstein–Barr virus. According to Duesberg, "the virus has since been found in every African in the country, was said to be a slow virus, now it's due to chromosomal transmutation plus a tumor, it's not infectious." A third example from his long list was cervical cancer. "In the 1970s [cervical cancer] was said to be due to herpes virus, in the 1980s [it was] due to papilloma virus. But 70–80% of the women carry the herpes virus or the papilloma virus, [yet] only 13,000 per year in this country develop cervical cancer. If you look at the 13,000, 70% have the virus and 30% don't. So you have to look for better causes for that and also it's not infectious" [71].

Our examination of the history of epidemiology demonstrates how scientists in current controversies construct and employ histories of medicine, technology, and science to support their arguments or to deconstruct opponent's arguments. This is more than a debating strategy. Constructing history is one means by which scientists reconstruct rules for verifying facts and findings; that is, constructing history is part of the self-vindication process. (This act is not at all limited to the participants in this controversy. We all do this, as exemplified by this text and discussed by recent works on the writing of history [73].)

Multiple factors, multiple practices

A second point of our telling the history of the redefinitions of Koch's postulates is to demonstrate epidemiologists' inclusion in every revision of a wider range of 'multiple factors' in making any conclusion regarding disease causation. Duesberg primarily selects historical cases where the germ theory has failed in the search for disease causation. While he might be correct in assuming that retrovirologists like Gallo believe(d) that HIV alone could cause AIDS, contemporary epidemiologists tend to believe in multifactorial disease models and therefore their acceptance of HIV as a cause of AIDS is based within an understanding of a "web of causes." "The major premise of the multifactorial model is . . . that a given disease may have a number of causes or antecedents, a combination of which may be needed to produce the disorder" [55, p. 51].

Another aspect of epidemiology's multifactorial approach, or what Evans calls the "complex interplay of agents, environmental, and host factors," is the issue of HIV's actions in different historical, geographical, and socio-cultural situations. For example, Winkelstein argues that HIV acts differently in different socio-geographical populations [74].

> Diseases behave differently in different populations. [Duesberg] doesn't seem to understand this. They behave differently in different circumstances . . . That's what epidemiology is all about. How do diseases behave in populations, and what factors influence them? . . . Duesberg [asks], for example, why is AIDS different in Africa? Why does it affect males and females in Africa and largely males in the United States? He says that doesn't make sense. It makes eminent sense. [D]iseases behave that way. If you introduce a sexually transmitted disease into a highly sexually active population like homosexual men in San Francisco, you'll get a huge epidemic. If you introduce a sexually transmitted disease into Africa where there very little homosexual activity but a huge amount of heterosexual mixing, where there are other diseases that cause ulceration and so forth and facilitate infection, you'll get a different disease. The disease will behave differently. The characteristic of diseases is that they don't behave the same in different

populations. That's what we are all about, epidemiologists, studying why disease is this way in this population and that way in that population. Same disease, but behaving in a totally different way because of all these factors that influence how diseases [behave]. That's why it's a science, that's why it's a big study [Winkelstein interview].

Winkelstein also presents the issue of migration and social movement as another example of epidemiology's concern with multiple condition involved in disease causation [75].

Duesberg comes to the conclusion [that HIV is an old virus because] its prevalence [hasn't changed] in the last 5 years... There simply isn't any evidence that it's an old virus. The evidence is entirely in the opposite direction: it's a new virus, at least in our population. It may have been an old virus in some other population. It may, for example, have been present in rural African populations at a very low level for eons. And then when in the past 25 years the populations, the villages were moved to the cities, the village cultures, the village society, the village ways of doing things changed. People mixed. And so you get a whole new set of diseases. We know that happened in Europe when the industrial revolution came. It changed the whole.. way people lived. So the diseases changed, because diseases are dynamic. That's what epidemiologists study, the dynamics. And those dynamics are affected by the cultures and the way people live and what they work at, whether they work in mines or whether they work in fields, farms, and so forth. All of those things interact to produce the distributions of disease [Winkelstein interview].

Changes and redefinitions

Perhaps more telling is a methodological point. Epidemiologists emphasize the importance of changing criteria to respond to the development of new technologies and concepts. For example, Evans argues that "all our concepts of causation are limited by the technology available to prove them and [by] our understanding of the pathogenesis and epidemiology of disease at the time of the investigation" [59, p. 250]. Change and the inclusion of new phenomena, information, instruments, research designs, and materials are standard operating procedure. In other words, change in technologies and substantive information is *standard procedure* in the history of epidemiology. Duesberg views this reliance on the latest available technology negatively. He claims that new technologies are used to define new 'diseases' with new causal agents, neither of which would exist except by way of the new technologies [39]. Evans views this reliance on changing technologies as part of the advance of science and knowledge.

Hacking notes that the self-vindicating character of styles of reasoning helps us to understand the "quasi-stability of science" [7]. Instead, our example of epidemiological practices emphasizes change as epidemiologists confront new diseases [76] and employ new technologies. We describe the construction of new practices and verification rules to maintain the epidemiological style as a self-authenticating system. Koch's postulates are still called Koch's postulates despite the fact that the practices, technologies, and elements involved in their application, definition, and interpretation have changed frequently during the last century.

The historical redefinitions of Koch's postulates parallel the historical redefinitions of epidemiology appearing in biomedical journals in 1930s, 1940s, 1950s, 1960s, and the 1970s. Epidemiologists are continually recrafting their craft as they engage new technologies, new disciplines of study, new diseases, new microbes, new population groups, new public policy agencies and politicized actors, etc. [77].

Mosaics

The 'mosaic' aspect of the epidemiological style of practice noted earlier in this paper is our fourth point. There are two parts to this issue. First, as we have noted, no one element or piece of information can adequately define the problem or the etiology. Only the composite view presents the viewer with a discernible picture or pattern. However, for Duesberg, each piece of information supporting the case against HIV should precisely fit with other pieces; otherwise researchers are building a house of cards. In contrast, epidemiologists and retrovirologists in AIDS research vindicate their conclusions through the *association and accumulation of ambiguous and incomplete data.* Analogies and examples, both historical and contemporary, are used to bridge gaps of information unavailable due to issues of ethics, privacy, economics, and organization. For example, Winkelstein states that Duesberg uses epidemiological statistics incorrectly because he lacks the background knowledge with which to interpret the data. Statistics are not to be taken literally; they are subject to interpretation.

Now, when he says that there's only... 120,000 cases of AIDS in Africa, he says that's not very much. Well, if you knew anything about the medical services in Africa, you would know that whatever number they give you is meaningless. Because they can't, there's no way of counting. Because there [are] no systems for accurate recording. Even in our country, where the recording is very good, and where we have all kinds of checks—for example we check death certificates against reports and then make estimates of the under reportings—we think that probably in this country the underestimation is probably in the neighborhood of 20%. So there are probably 20% more cases than are getting reported. And that's going to vary. In some places it's going to be 40% and an in some places it's going to be 2% and so forth [Winkelstein interview].

The second 'mosaic' aspect explicit in the above definitions of epidemiology and in histories of AIDS epidemiology is that AIDS researchers *associate and accumulate information and technologies (material and procedural) from many different lines of work* to construct and vindicate their conclusions about disease etiology. AIDS researchers rely on the expertises of retrovirology, immunology, and cell biology to define the disease-causing agents and activities of HIV as they operate in human organisms. They rely on the expertise of medical practices to make reliable dignoses of symptoms and illnesses. They rely on sociology and anthropology to define relevant risk

population groups and their cultural and behavioral practices. In other words, they weave *a web of practices* as well as *a web of causes*.

Indeed, a process of selecting and combining different information and technologies in AIDS epidemiology gave epidemiologists reason to propose in 1983 that AIDS was caused by an infectious agent—that is, *before* retrovirologists introduced HIV as the causal agent. Oppenheimer argues that between 1981 and 1983, epidemiological studies of AIDS concluded that AIDS was caused by multiple 'lifestyle' factors that predisposed patients to immune dysfunction and infections. These 'lifestyle' factors included homosexual sexual practices (e.g. many partners, anal contact) and use of recreational drugs (e.g. amyl nitrites or 'poppers'). Oppenheimer notes that epidemiological practices were criticized for reproducing negative cultural views of homosexuals and IV drug users in their definition of the disease (e.g. Gay Related Immunodeficiency or GRID was the original name given to the disorder) and the procedures for its surveillance. "In the face of a fatal disorder of unknown origin and indefinite proportions, epidemiology offered a set of procedures (for example, case definition, verification, and count) that swiftly generated results and then authenticated them, giving the public a sense of definite progress" [55a, p. 52]. However, Oppenheimer also notes that epidemiologists and the CDC rejected the lifestyle hypothesis and concluded that an infectious agent was the necessary cause of AIDS in 1983, also on the basis of epidemiological studies [78]. Further, although epidemiologists accepted the 1984 designation by retrovirologists of HIV as the infectious agent, epidemiologists have maintained a multifactorial approach to the disease.

> The possible role of cofactors testifies to the terrible complexity of HIV infection and justifies the reluctance of epidemiologists to reduce AIDS and related conditions to an agent-host phenomenon. Epidemiological researchers have consistently held up the possibility of nonviral factors to the 'bench' scientists. Since 1981 they have rooted biological or clinical events in the matrices of human behavior and social experience. In one study of the role of cofactors in HIV infection, the authors put the epidemiologists' position quite well. Citing the viral etiology common to all patients with AIDS, they stressed the multiple determinants probably responsible for HIV infection and disease progression, including cultural differences, the presence of other endemic illnesses, and host and viral genetic factors. Their position reaffirms the multifactorial model as central to an understanding of HIV infection and to its control [55, p. 68].

According to Oppenheimer, this multifactorial framework has already benefited, and will continue to benefit, efforts to contain and control the spread of AIDS while everyone waits for elusive cures and vaccines. Further, how these technologies and information are selected and associated to construct explanations and justifications for hypotheses is also open to scrutiny.

> [B]y defining HIV infection as a multifactorial phenomenon, with both behavioral and microbial determinants, epidemiologists offered the possibility of primary prevention, a traditional epidemiological response to infectious and chronic disease. Epidemiologists, in effect, established the basis for an effective public health campaign and . . . helped make AIDS a concern of policymakers and the public [55a, p. 76].

However, other writers are more critical than Oppenheimer about the quality of information provided on AIDS. For example, several writers criticize the anthropological and sociological information about African AIDS and HIV infection cases and statistics generated by Western studies. Packard and Epstein question anthropologist and physician Daniel Hrdy's view (and also expressed by Winkelstein above) about the relationship between African migration patterns, loss of "traditional restraints," greater sexual promiscuity, and HIV transmission [79]. They argue that Hrdy's "image of the 'detribalized' African, the bane of colonial urban authorities, was a central image in earlier discussions of black susceptibility to TB and syphilis. This image, which was fairly well excised from social science discussions in the 1970s, was being resurrected to explain the frequency of heterosexual transmission of HIV and Africans in the 1980s." This view, they argue, focuses attention on sexual promiscuity and migration patterns and "deflects attention from other co-factors that may be as important for the heterosexual transmission of AIDS in Africa as frequency of sexual contacts," such as background infections by other microbes and malnutrition [79, p. 357].

Treichler similarly argues that statistics on African AIDS and HIV infection must be understood within the complex, heteroglossic situations of their production and consumption [1]. She cites flaws in blood testing and other diagnostic procedures in African 'AIDS' cases that are then translated into flawed statistics. She points to cultural practices taken out of the local cultural context and turned into rumors and fantasies "fueled by historically entrenched myths of the exotic" [80]. These numbers then take on lives of their own as they are deployed in local and global political discourses and agendas.

In the meantime, whether or not categories are nonequivalent, whether or not numbers are exact references, HIV-AIDS researchers are weaving connections between them to vindicate the theory that HIV causes AIDS. In the process, they are defining the disease.

Defining the disease

Diagnosis, definition, and classification of disease are intricately interwoven in medicine. Fagot-Largeault [56] tells us that contemporary epidemiology faces problems similar to those faced by researchers who studied the causes of death in the nineteenth century. That is, epidemiology was and is limited by the quality of the diagnoses made by physicians. In contrast, officially problematic diagnosis has not been considered to be a major problem by

AIDS epidemiologists in the First World, because physicians are using the formal CDC definition of AIDS to diagnose AIDS cases [81].

According to Duesberg, however, *diagnosis* is problematic. He argues that illnesses are currently diagnosed according to the definition of AIDS as HIV seroposivity and the clinical manifestation of one of twenty-five conventional diseases, including tuberculosis, Kaposi's sarcoma, *Pneumocystis carinii* pneumonia, etc. (The CDC currently lists 25 illnesses as symptoms of AIDS.) Duesberg's argument is that, given this definition, only incidents of the disease in HIV-positive patients are identified as cases of AIDS, that HIV-negative cases are not classified as AIDS, and that the circular definition eliminates any possibility of finding the 'real' cause of AIDS.

> The definition of AIDS, that's all there is to it. It's a combination of one or several of twenty-five old diseases in the presence of antibody to virus. Example: Tuberculosis with HIV is AIDS, without it is tuberculosis. Dementia with HIV is AIDS; without [HIV], you're just stupid. It sounds funny, but that is the definition of AIDS. That's exactly what it is. There's no other definition of AIDS. It's all diseases in the presence of HIV. In its absence, they get their own names [Duesberg, Berkeley Statistics Department colloquium, May 1992].

However, the official Centers for Disease Control's AIDS *definition* is more complex and problematic than Duesberg indicates. As we have noted, the CDC's definition (until 1 January, 1993) did not require a positive HIV test to qualify as a case of AIDS. Moreover, the official CDC definition has been revised numerous times since its original nomenclature as Gay Related Immunodeficiency [15, 55a]. For example, advocates have long called for the designation of HIV infection as a disease, separate from 'full blown' AIDS. The National Academy of Sciences committee concluded that "HIV infection itself should be considered a disease ... [I]t is now clear that AIDS is end-stage HIV infection" [55b, p. 78]. However, while the CDC does not designate HIV-infection as a disease, they do track HIV-infected people.

In 1991 the CDC proposed another change in the definition of AIDS to reflect another disease marker, low CD4 cell count (that is, a CD4 count below 200 cells per mm^3 of blood, which is about 80% below the population mean) [82]. However, the Social Security Administration objected to this planned redefinition, because it would have had to bear part of the costs incurred by the increases in AIDS cases under the new definition. (Note here that we had two arms of the federal bureaucracy fighting over how to define the same object.) But the CDC did not give up. In Fall 1992, they successfully reintroduced their proposed revision to include HIV-infected people with CD4 count below 200 in the definitions of AIDS which took effect on 1 January 1993.

Meanwhile, other groups lobbied for other revisions in the definition. For example, women health activists called for the inclusion of other illnesses (e.g. aggressive cervical cancer, pelvic inflammatory disease, and yeast infections) suffered by women infected with HIV in the list of AIDS-symptomatic diseases. The CDC agreed in October 1992 to add invasive cancer of the cervix to its new definition which took effect on 1 January 1993. Patients, doctors, and AIDS activists also succeeded in pressuring the CDC to include pulmonary tuberculosis and two or more episodes of bacterial pneumonia to the new definition of AIDS.

These are just a few examples of the different parties fighting to participate in defining the disease. With the inclusion of these new disease categories in the definition of AIDS, tens of thousands new AIDS cases were predicted to be added to the official AIDS count [83].

While Duesberg raises an important question about whether diagnosis simply mirrors CDC's official definition, his simplification of the issues of definition obscure the value of his question. Moreover, Duesberg's view of the AIDS diagnosis masks the consideration given to the patient's immune system in both official definitions and local diagnoses. AIDS epidemiologists such as Winkelstein argue that Duesberg's criticisms ignore the 'real' disease, that is, a deficient immune system which in turn causes other diseases to thrive.

> [A disease] can be caused by the same agent and have different outcomes ... [Duesberg] says the fact that we have all these different diseases doesn't make sense. Well, we really only have one disease. We have an underlying immune deficiency, and that's what the disease is, immune deficiency. These other diseases are ... consequences of immune deficiency. If you don't have any immunity, you can get a lot of different diseases [Winkelstein interview].

Nevertheless, Duesberg's critique points our attention to the fact that the definition of the disease itself is a much debated product of negotiations among many parties, concerns, and factors. According to Duesberg, virologists and epidemiologists have constructed a self-fulfilling definition. In contrast, we argue that the definition of AIDS at any moment is the outcome of negotiations among virologists, immunologists, cell counts, HIV, the economics of medical care, patient activist groups, the CDC, the Social Security Administration, the Department of Health and Human Services, the World Health Organization (WHO), epidemiology and its history, and so on. AIDS diagnoses are similarly multi-dimensional and therefore fraught with the difficulties presented by the indivisibility of diagnosis, definition, and classification of AIDS cases.

Consequences

In the end, it is the consequences of different positions on this HIV-AIDS controversy that matter. The official definition of AIDS and the theory that HIV causes AIDS are currently being used to design public health policy prevention measures, treatment and prevention (vaccination) research and protocols,

and in evidential testimony in judicial decisions (e.g. in assault trials). Duesberg argues that the outcomes of these measures will be harmful, because HIV does not cause AIDS. For example, he claims that AZT is killing people rather than curing them. He also argues that funds should be allocated not to research on AZT and other HIV-killing drugs but instead to explorations of other means for controlling the disease. More dangerous to the ears of AIDS researchers is his claim that AIDS is not an infectious disease. In their view, this statement undermines all the prevention and control measures currently underway and is tantamount to telling people to cause harm to themselves—that is, that Duesberg is telling people that they will not contract AIDS from practicing unsafe sex or from sharing dirty needles.

CONCLUSION

The hypothesis that HIV causes AIDS is accepted as fact by most AIDS researchers, physicians, patients, public health organizations, and governments involved in AIDS research, prevention, and treatment [84]. Peter Duesberg dissents from this view and has attempted to enroll others, including epidemiolgical data, retrovirological data, cell biology results, the news media, and researchers, in his dissent. We discuss how he selects, amasses, juxtaposes, and sometimes transforms each set of 'evidences' from different worlds of practice, much to the dismay of the practitioners whose data he interprets in ways they consider to be inappropriate. Duesberg here assembles a postmodern collage or pastiche from a plurality of discourses to argue his case.

We use Duesberg's criticisms and the responses to his dissent to attempt to explore the politics and practices of epidemiological science. We call its politics and practices 'a style of practice.' Our styles of practice are a modification of Hacking's styles of scientific reasoning [7]. Styles of practice stress the historically located collective efforts of scientists, technicians, administrators, institutions, and various 'publics' as they build and sustain ways of knowing. We use the term practice rather than reasoning to emphasize that reasoning cannot be separated from practice in our approach.

We view knowledges as situated and collectively constructed. As social scientists studying knowledge construction and verification, we ask: what are the relevant situations, actors, actions, interactions, and outcomes. Following this approach, we have used Duesberg's dissent to gain access to epidemiological arguments and practices supporting the thesis that HIV causes AIDS. We have examined his criticisms and epidemiologists' answers to his criticisms to explore and define some elements of an 'epidemiological' style of practice that researchers have used in the last ten years to 'verify' the theory that HIV causes AIDS. Our study of practices used in epidemiology, in contrast to the philosopher's focus on ways of thinking or cognitive styles, has provided us with some understanding of the rules of verification used by those who support the HIV causal agent theory. It has also provided us with an example of scientists' construction, deconstruction, and reconstruction of historical continuities in their efforts to support or criticize scientific theories.

The epidemiological style of practice entails constructing a mosaic of information, materials, technologies, and abstractions produced by diverse worlds of practice. Epidemiological information comes in many forms from many social worlds and from historical studies of other diseases. This means that epidemiologists rely on the expertise of these diverse contemporary and historical worlds of practice. Since information is inseparable from its means and situations of production, the mosaics constructed by AIDS researchers might include incongruities, incompleteness, untranslatable differences, and errors in information. However, epidemiologists argue that these problems are unavoidable given the objects they study, that is, diseases in populations.

Most important for the debate about whether HIV causes AIDS are the recurrent revisions of rules for establishing disease causation. Epidemiologists present their current style of practice as the result of ongoing efforts to construct rules for judging statements that work to overcome or adjust for the problems discussed above. Using their various revisions of Koch's postulates as our example, we demonstrate that epidemiologists refer to histories of these practices as they have changed over time and through experiences with different microbes and diseases to legitimize and texturize their current practices. For epidemiologists, this is a 'natural' updating of the field as new problems occur and new technologies and materials are introduced and taken up. For our dissenter Duesberg, this is an example of moving the goal posts in the middle of the ball game. For us, understanding that these rules are historical objects constructed and changed through new situations (new diseases, new microbes, new environments, new technologies, new concepts) tells us that this style of scientific practice is a self-authenticating system, (re)constructed in context. Like other biomedical (and to some extent all) sciences, epidemiology is a complex set of practices that mediate the relationship between clinical and field observation and theory construction and verification. This set of practices, and therefore this relationship between observation and theory, is dialectical/undetermined/underdetermined and under continual (re)construction. Nevertheless, it is a system for establishing facts where observations and the rules for interpreting observations are co-produced to enable researchers to construct what Dewey called "warranted assertions" [85].

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44. See Boltanski L. and Thevenot L. *De La Justification. Les Economies de la Grandeur*. Gallimard (NRF Essais), 1991, for an architecture for discussing actors' appeals to different regimes of action to justify their actions. See Dodier N. Agir Dans Plusieurs Mondes. *Critique* 529–530, 427–458, 1991, for a summary and review of Boltanski and Thevenot's work.
45. See [7, pp. 16–19]. Hacking does not include branches of logic in their styles of reasoning, because deduction, induction, and abduction are practiced by all people and are not peculiar to scientific styles of reasoning.
46. Our approach differs from S. Cole's recent discussion of the role of 'truth' (Cole S. *Making Science: Between Nature and Society*. Harvard University Press, Cambridge, MA, 1992). Cole views socially constructed knowledge as ephemeral. In contrast, our approach to the issue of 'truth' can be summarized in the statement that "just because something is constructed does not mean that it is not real." Instead of continuing the persistent efforts to "pin down" truth and reality, we discuss what is observable by sociologists/anthropologists. This means that we examine the practices and activities of scientists in their efforts to construct facts, rather than try to adjudicate the issue of truth. I discuss this approach elsewhere in Fujimura J. *Crafting Science*. Harvard University Press, Cambridge, MA, in press.
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51. 'Nude' mice are specifically bred to have a minimal immune system for experimental studies. Their name symbolizes their lack of hair, lost in the breeding process. They cannot survive outside of carefully controlled laboratory conditions.
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61. Morris J. N. Uses of epidemiology. *Br. Med. J.* **2,** 395–401, 1955.
62. See also Abramson J. H. RE: definitions of epidemiology. Letter to the Editor. *Am. J. Epidemiol.* **109,** 99–102, 1979; Aycock L. W. and Russell, F. F. The functions of hypotheses in epidemiology. *Am. J. med. Sci.* **206,** 399–408, 1943; Bundesen H. N. and Hedrich A. W. Method for early detection of epidemic trends. *Am. J. publ. Hlth* **15,** 289 296, 1925; Chave S. P. W. Henry Whitehead and cholera in Broad Street. *Med. History* **2,** 92 109, 1958; Crookshank F. G. First principles: and epidemiology. *Proc. R. Soc. Med.* **13,** 159–184, 1919–1920; Frerichs R. R. and Neutra R. Definitions of epidemiology. To the Editor. *Am. J. Epidemiol.* **18,** 74 75, 1978; Lillienfield D. Definitions of epidemiology. *Am. J. of Epidemiol.* **107,** 87–90, 1978; Rich H. More on definitions of epidemiology. To the Editor. *Am. J. Epidemiol.* **109,** 102, 1979; Stallybrass C. O. *The Principles of Epidemiology and the Process of Infection*. George Routledge & Son, London, 1931; Taylor I. and Knowelden J. *Principles of Epidemiology*. Little, Brown and Company, Boston, 1957. See also a series of editorials in the *Am. J. publ. Hlth* on the topics of "what and who is an epidemiologist?" and "what is epidemiology?" (April, June, July, August, September, November 1942, and June 1948).
63. Jenicek M. and Cleroux R. *Epidemiologie clinique*. Edisem, Clinimetric, Montreal, et Maloine, Paris, 1985.
64. Since this publication, HIV has been found in individuals not included in the designated high risk populations.
65. Strauss A. *Continual Permutations of Action*. Aldine de Gruyter, Hawthorne, NY, 1993; Suczek B. and Fagerhaugh S. AIDS and outreach work. In *Social Organization and Social Process* (Edited by Maines D. R.), pp. 159–174. Aldine de Gruyter, New York, 1991; Wiener C. Arenas and careers: the complex interweaving of personal and organization destiny. In *Social Organization and Social Process* (Edited by Maines D. R.), pp. 175–188. Aldine de Gruyter, New York, 1991.
66. However, as Lewontin has recently noted, medical cures have rarely come from understandings of the causal processes. Lewontin R. The dream of the human genome. *N.Y. Rev. Books* **39,** 30–40 (p. 35), 1992.
67. Efforts to construct a suitable animal model continue. For example, scientists at the University of Washington's Regional Primate Center announced at the end of June 1992 that they had created a potential animal model for testing the HIV hypothesis. They managed to infect pigtail macaques with the HIV (Agy M. B., Frumkin L. R., Corey L., Coombs R. W., Wolinsky S. M., Koehler J., Morton W. R., and Katze M. G. Infection of *Macaca nemestrina* by Human Immunodeficiency Virus Type-1. *Science* **257,** 103–106, 1992). However, their work has not been reproduced and that hope is fading for this model (Brown P. Hopes fade on perfect animal model for AIDS. *New Scientist* **8,** 1992). New announcements of potential animal models have appeared since then in *Science*, but none has yet satisfied AIDS researchers.
68. Hammonds E. The search for perfect control: the social history of diphtheria, 1880–1930. Ph-D. Dissertation, Harvard University, Cambridge, MA, 1993.
69. Gajdusek D. C. Subacute spongiform encephalopathies: transmissible cerebral amyloidoses caused by unconventional viruses. In *Virology* (Edited by Fields B. N., Knipe D. M. *et al.*), pp. 2289–2324. Raven Press, New York, 1990. Kuru had previously been thought to be a sorcery-caused illness and a psycho-somatic disease [Lindenbaum S. *Kuru Sorcery*. Mayfield, Palo Alto, CA, 1979; cf. Anderson W. Securing a brain: the contested meanings of Kuru. In *So Human a Brain* (Edited by Harrington A.), pp. 193–203. Birkhauser, Anderson, Boston, 1992)].
70. Beginning in the 1950s, epidemiologists also began to discuss methods for understanding causation in chronic diseases. With the exception of a few rare genetic, single-gene diseases, the research on non-infectious chronic disease faced the problem of showing the specificity of presumed causal factors. Even here, a disease like sickle cell anemia, a single-gene disease, exhibits a range of clinical manifestations, depending on other host and environmental factors (Duster T. *Eugenics Through the Back Door*. Routledge, London, 1990). Fagot-Largeault [56] discusses the use of statistics in determining chronic disease causation.
71. Duesberg P. AIDS: infectious or not? Statistics Colloquium, Department of Statistics, University of California, Berkeley, 1992.
72. Duesberg P. Virus hunting and the scientific method. *Science* **251,** 724, 1991.
73. For example, Borofsky R. *Making History: Pukapukan and Anthropological Constructions of Knowledge*. Cambridge University Press, New York, 1987; Burke P. (Ed.) *New Perspectives on Historical Writing*. Pennsylvania State University Press, University Park, PA, 1991 [1986]; Dening G. *History's Anthropology: The Death of William Gooch*. (ASAO special publications; number 2). University Press of America, Lanham, MD.
74. At another level, Treichler [1] argues that *information about* how HIV infection and AIDS behaves differs by geographical and social situations. Therefore, she argues, numbers and statistics should be taken narratively and not literally.

75. However, see the section on 'Mosaics' for a contradiction of Winkelstein's African migration explanation.
76. However, see Grmek [15] for a discussion about whether AIDS is really a new disease.
77. For good examples of definitions and redefinitions, see [60, p. 38]; [61, pp. 396, 399]; [62 (Aycock and Russell), p. 400].
78. See, for example, Marmor M., Friedman-Kein A. E., Laubenstein L., Byrum R. D., William D. C., D'Onofrio S. and Dubin N. Risk factors for Kaposi's sarcoma in homosexual men. *Lancet* **1**, 1083–1087, 1982; Jaffe H. W., Choi K., Thomas P. A. *et al.* National case-control study of Kaposi's Sarcoma and *Pneumocystis Carinii* pneumonia in homosexual men: Part I, epidemiologic results. *Annls internal Med.* **99**, 145–151, 1983.
79. Packard R. M. and Epstein P. Medical research on AIDS in Africa: a historical perspective. In *AIDS. The Making of A Chronic Disease* (Edited by Fee E. and Fox D. M.), pp. 346–376. University of California Press, Berkeley, 1992.
80. See especially Treichler's [1] footnote 33 for references to literature on problems with information on African AIDS and HIV infection.
81. Nevertheless, mistakes are made in diagnostic procedures in the Western world. Winkelstein tells a story about a study on polymerase chain reaction (PCR), a sensitive procedure for testing for the presence of HIV, as employed by five different research laboratories. The labs agreed to participate in the study and knew in advance that the results would be published. Yet, at least 40 clerical mistakes were made: "checking the wrong specimens, calling them positive when they were negative, getting the numbers mixed up . . . You cannot believe how easy it is to get these things mixed up" [Winkelstein interview 1992].
82. CD4 refers to a protein that sits on the surface of the T-4 lymphocytes (immune system cells). This protein is used as a marker for counting T-4 cells, since immunologists have designed efficient and standardized methods for identifying the protein (Cambrosio A. and Keating P. A matter of FACS: constituting novel entities in immunology. *Med. Anthropol. Q.* **6**, 362–384, 1992). The molecular biological argument is that the CD4 protein chemically 'attracts' a protein on the surface of the HIV, allowing the virus to then move its genetic material into the cell and make it reproduce viral copies which in turn attacks enough T-cells to kill the host.
83. Altman 1992.
84. Cases of AIDS-like diseases without the presence of HIV have been the recent big news in AIDS research. Although such cases have existed for some time [35], they have been largely ignored. Many researchers have assumed that the absence of HIV was due to poor methods for detecting the presence of HIV. However, current PCR methods are sensitive enough to make this an unlikely explanation for the absence of HIV in new AIDS cases. Some researchers have argued that these cases are caused by a new virus, while others argue that this is an entirely new disease. Others like Anthony Fauci, head of NIAID, are skeptical about these claims. At this time, there are no answers. CDC has instituted a new category named "idiopathic CD4+ T-lymphocytopenia" (ICL) for the 111 cases (as of May 1993) they judge to fit their criteria. The criteria CDC uses to define this category include: two separate CD4 tests below 300 cells per mm^3 of blood . . . or CD4s less than 20% of the total lymphocytes; no known causes of immunodeficiency or therapy that could be deplete T cells . . . ; and . . . no evidence of infection by the AIDS viruses, HIV-1 or HIV-2" [p. 1032 in Cohen J. "Mystery" virus meets the skeptics. *Science* **257**, 1032–1034, 1992].
85. Dewey J. *Logic: The Theory of Inquiry*, p. 4. Henry Holt, New York, 1938.