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The Cognitive Closure of Science
Case Study: the Discourse about the Etiology of AIDS, 1981–1986

Abstract: As the sociology of scientific knowledge has revealed, research fields may frequently maintain or legitimize hypotheses independently or in the absence of experimental data or other empirical evidence constituting conclusive scientific proof in accordance with declared methodological standards. This essay aims to show certain of the mechanisms and social factors that allow scientific discourse to function as a self-referential system, i.e., in an autonomous manner in regards to the border conditions of empirical experience, as described by W. Quine. I particularly concentrate here on how the organization of scientific work in selected disciplines can result in the local findings of individual laboratories being quickly transformed into unrevisable facts (black boxes). The phenomenon of the self-reference of scientific discourse is well illustrated by the case of the debate on the cause of AIDS. This discourse was so configured that by referring to one another and by theoretical imputation researchers caused the hypothesis on the causal relation between HIV and AIDS to begin to be accepted as an indisputable fact, even though the corroborating evidence had not appeared in the meantime.

Keywords: black box, scientific discourse, HIV/AIDS, sociology of scientific knowledge, self-referential system

Introduction

It is traditionally considered that objective reality—understood as something external and independent of the research process—plays a part in closing scientific controversies and shaping our knowledge. Many contemporary research trends in the sociology of scientific knowledge (SSK) and in science and technology studies (STS), however, display the limitations or inadequacy of such an epistemological approach. They suggest that scientific knowledge is characterized by "limited empirical sensitivity." Often science turns out to be cognitively closed to the "external" world. In this context the category of *autopoiesis* can be recalled [see Luhmann 1990, 1995; see also Maturana, Varela 1980]. Here, I understand *autopoiesis* as the self-reference and self-legitimization of discourses, i.e., the point of reference for knowledge generated by the social system is not the "external" world, but the cognitive system itself—to a large degree it is able to legitimize knowledge independently of what could be described as empirical input. Metaphorically speaking, social systems are "encysted" in such a manner that the cognitive process takes place in a sort of cocoon, which acts as "protection" against the influences of the environment. These

influences do not reveal themselves directly, but are always socially filtered [Zybertowicz 1995: 118].

The main aim of this article is to describe the processes and factors that produce science's cognitive closure to the world. What is of interest is the manner in which scientific knowledge may legitimize itself independently of the results of observations and experiments. The *autopoiesis* of cognitive systems will be treated here not as a theoretical assumption, but as a category requiring empirical exemplification. The basic perspective of the essay is that of the sociology of scientific knowledge (further SSK) [see Knorr-Cetina, Mulkay (ed.) 1983]. This discipline was initiated by the Strong Programme [Bloor 1991: 7] of the Edinburgh School [see Barnes, Bloor, Henry 1996], which postulated that sociology should explain not only the context of science (as had been done previously) but also its content. The Edinburgh School paved the way for successive waves of sociological studies of science, such as: (1) the ethnography of laboratory practice [Knorr-Cetina 1981, 1983, 1999; Lynch 1985] and the anthropology of science [Latour 1987; Latour, Woolgar 1979], (2) the sociological analysis of scientific discourse [Gilbert, Mulkay 1984], and (3) the Bath School's Empirical Programme of Relativism (EPOR) [H. Collins 1983, 1985; Collins, Pinch 1998a, 1998b].

In the first part of the text I reconstruct the processes and factors that often cause scientific knowledge to be constituted and to persist independently of objective reality, exterior influences on science, or "empirical" data. The second part of the essay is an extensive case study of a self-legitimizing scientific hypothesis. It pertains to the debate over the cause of AIDS in the years 1982–1986. As we shall see, the causal relation between HIV and AIDS, which is universally accepted today, was constituted in the absence of experiments, observations, or other premises recognized as definitive scientific proofs by the standards of the discourse itself.

The SSK Perspective on the Cognitive Closure of Scientific Discourses

The Duhem-Quine Thesis

Let us begin by referring to the Duhem-Quine thesis, known also as the underdetermination thesis. Willard Quine claimed that our scientific knowledge is like a field of force whose boundary conditions are experience. The field as a whole is so undetermined by these boundary conditions that there is much latitude of choice as to what statements to reevaluate in the light of any single contrary experience. Particular elements of the field can be readjusted in order to make them fit each other and eliminate conflicts [cf. Quine 1951: 42–43]. According to Pierre Duhem, theoretical explanations and experience can be made to fit each other not only by changing the logical value of individual claims or the rules of logic. As he showed using the example of astronomy, when observation is incompatible with theory, one can revise (1) the astronomical theory, (2) the law of the propagation of light in space, or (3) the theory explaining how the instrument (in this case, the telescope) works [cf. Duhem 1905].

This leads to a situation where experience and theory are made to fit each other and thus create a certain closed, mutually confirming, whole [cf. Hacking 1992; cf. robust fit, Pickering 1995]. Ian Hacking [1992] showed that experience can be fitted to theory through a change in the theory, an auxiliary hypothesis concerning the functioning of the instrument, and/or the rebuilding of the instrument itself or possibly a change in the way it is utilized.

The underdetermination of scientific knowledge by the objective world does not lead to the cognitive closure of systems. But this observation makes us aware of how long scientists can stick to their position in the face of the most varied evidence and how much freedom they have in adapting experience and the theory at their disposal. The empirical experience is inseparably connected with scientific theory—there is no pure experience, because all experience is categorized within a particular theoretical framework and the use of a scientific instrument is based on a number of tacit or explicit assumptions.

Black Boxes and the Costs of Replication of Experiments

Much more interesting from the perspective of our analysis are the mechanisms associated with the "economy" of scientific research and the costs of replication of experiments. Among the elements ensuring the stability of scientific findings are the unusually high costs of certain scientific experiments. The replication of research is frequently so costly that researchers will not undertake it. (An exception is a situation where the results of a given experiment do not agree with other elements of the discourse, i.e., with the results of competing research teams or theoretical predictions). Let us consider the following example of research on the thyrotropin-releasing hormone (TRH) conducted by the Salk Institute [see Latour, Woolgar 1979; cf. Hacking 1999: 175]. At the time, there was not a large-enough quantity of TRH samples available to be used for laboratory analysis. In order to get the one microgram of the substance that the Salk Institute needed to perform its original research, five tons of pigs' brains were transported on ice from a slaughterhouse in Chicago to the laboratory in San Diego and samples of TRH were extracted. No laboratory could afford an analogous enterprise exclusively for the purpose of confirming the results achieved by the Salk Institute. These types of expenditures are justified only in the case of pioneering work. Obviously, not all natural science disciplines require similar expenditures. Even so, for scientists the rational choice would seem to be to continue and develop research work that already exists and not to contest or undermine other work, with the possibility of becoming embroiled in long-lasting controversy. It is more profitable to treat those results as a kind of black box.

The term "black box," which is taken from cybernetics, functions in SSK as something more than a loose metaphor. In this paper, by "black box" I mean every object that constitutes a stable construct [see Latour 1987: 1–3]. A black box may be (1) an effectively operating technological artifact, (2) a standardized, repeatable experiment, or (3) an established scientific claim constituting an entry point for further inquiry. The essence of black boxes is that researchers treat them as unproblematic

entities and use them in their research work, "building upon them" successive experiments, technology, or conceptions. Ideas, experiments, observations, or conceptions treated as a black box can become an element of a broader theoretical framework or be treated as an auxiliary hypothesis explaining how a particular instrument works; equipment considered to be a black box can be used as an element of a larger technological system or a complex experimental set. In many instances, the process of closing a scientific black box consists in rejecting knowledge about the process of its construction [cf. Latour 1987: 6–7]. A black box can be defined in categories of the costs of its potential opening (problematizing) [Sojak 2004: 238–244]. Theoretically, there is always the possibility of opening a black box, but most often no one undertakes such a task because of the high costs. Black boxes are stabilized not only by various kinds of interests, convictions, and practices, but primarily by the process of building successive layers or levels of black boxes; attempting to open the bottom box would entail dealing with the whole stack of boxes built on top of it [cf. Callon 1991].

Let us return to the problem of replication. The fundamental difficulty associated with replication consists in the fact that it requires high experimental skills. Often the only method for ensuring the reproducibility or standardization of experiments is the transfer of tacit knowledge.[1] Confirming the experimental results of another laboratory or reproducing a non-standard device is not a trivial activity. It frequently requires the researchers to repeat nearly the entire cognitive process, and it sometimes leads to a situation where a satisfactory level of standardization for a given experiment can be achieved only by engaging the same highly-skilled person to perform the task. The differences in experimental fluency and "style" between laboratory researchers and technicians can be so large that they generate unacceptable discrepancies in the results of experiments [see Lynch 1985: 67–68]. Furthermore, replication is not only costly (it absorbs scarce research resources), but above all, time-consuming (it requires recreating the resources of tacit knowledge). Primarily, however, a research team must take into account the activities of other representatives of the research field: If a given laboratory were to concentrate on the replication of a particular, uncontroversial research result, other scientific centers could, during the same period, carry their projects forward, gaining the lead in access to grants and other sources of research funds. To state the matter in slightly different words, researchers considering the replication of an experiment or prototype must take into account the "costs" of potentially lost benefits (cf. opportunity costs).[2]

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[1] Harry Collins [1974, 1985: 51–78] analyzed attempts to replicate a certain type of laser (a TEA laser) by a research team. The device had been developed earlier in another research facility. The team he observed had at its disposal a detailed description and full documentation of the laser. These were insufficient, however. During the course of work a series of difficulties appeared that made reproduction of this comparatively simple artifact impossible. Replication was achieved only after the team was joined by a person who had participated in the work of the laboratory that had first constructed the device: This person had the skill-based, embodied know-how necessary to build and operate the TEA laser. The significance of tacit knowledge in scientific work was revealed by a sociological study of contemporary laboratories engaged in designing and constructing thermonuclear arms [MacKenzi, Spinardi 1995].

[2] This type of analysis in categories of cost calculations and of market-type mechanisms, presenting the image of science without romance, can be found, in the works of Pierre Bourdieu [1988], Philip Kitcher [1993, 2001] and Jesús Zamora-Bonilla [see: Ferreira, Zamora-Bonilla 2006; Zamora-Bonilla 2006, 2010].

The Organization of Scientific Research and the Closure of Black Boxes

In connection with Stephan Fuch's theory of scientific knowledge production [Fuchs 1992, 1993], an organizational model of science can be formulated that appears valid for many contemporary research fields, especially in the case of research fronts. The term "research front" refers to a field in which the most expensive and scarcest research resources and the most talented scientists are concentrated. These fields are characterized by a high degree of task uncertainty: the methodology and research techniques have yet to be worked out, the research object under study is just being constituted, and the scientists are functioning in conditions of great uncertainty as to the possibility of achieving their research aims. Above all, however, we are dealing here with a high degree of social control and mutual dependence of researchers.

Researchers functioning within the framework of research fronts are subjected to strong social control from the rest of their scientific community, where: (1) conducting experimental research within the community and writing credible reports requires access to expensive scientific resources (instruments, rare samples); (2) control over resources is at the disposal of co-researchers and the institutions financing and overseeing the research (reviewers of grant applications are most often other researchers); and (3) cognitive success is dependent on whether scientific theses are positively received and further developed or applied by the remaining representatives of a given discipline (they can reproduce it in their work, making it more stable). The more expensive or difficult to access the research resources are, the greater the extent of mutual control that researchers have over each other's work [cf. Fuchs 1992, 1993]. A high degree of reciprocal social control imposes methodological and theoretical discipline. A researcher's failure to recognize and join the main trend of research (for instance, by formulating an excessively innovative thesis that does not correspond with the categories and expectations of the reviewer) could meet with a denial of further research financing, which could hinder access to other research resources (a vicious circle can be created—the lack of funds for research leads to a lack of credible scientific results, which leads to difficulties in acquiring funds for research). At the same time, research concentrated on new types of problems is related to access to greater advantages and scientific capital, i.e., higher scientific prestige, which can facilitate the obtaining of grants, publication in prestigious journals, access to advanced scientific equipment, or personnel with the highest qualifications. In conditions of high mutual dependence, any scientific disputes are relatively quickly resolved by cutting off the resources necessary for research; rogue scientists, apostate trends or alternative scientific schools are most often eliminated. Attempts to break out of the paradigm preclude the realization of private research aspirations (which—it can be supposed—are rather high in very dynamic research areas).

In conditions of high mutual dependency, the winning strategy is to develop previous research effects and not to attempt any scientific coup or undermining of the achievements of others. Replicating test results or becoming embroiled in controversy also appears to be unprofitable. In pursuing the winning strategy, scientists build their work on the research of other researchers, thus effectively turning previous results

into black boxes. Cognitive effort becomes channeled. Researchers focus on resolving successive problems; they fill in the gaps the same paradigm and when they have finished with it they move on to other research fields, where as yet untapped resources and sources of credit await them. Randall Collins calls this model of scientific research "high-consensus, rapid-discovery science" [R. Collins 1994, 1998: 523–569]. Of course, not all disciplines function in accordance with this model. For example, some disciplines, particularly social sciences and humanities are not heavily reliant on rare research resources, which would enforce the researchers' dependency; the result is that controversial research is not as effectively closed or converted into a black box as in the natural sciences. In such conditions, incommensurable, parallel paradigms can develop, because researchers do not have the means of social control by which to discipline one another.

Even in the case of scientific disciplines requiring rare and costly research resources, the above organizational mechanisms sometimes fail and expensive controversies arise. What is important, however, is that rapid-discovery science acts in such a manner that the knowledge produced is rapidly made unverifiable. The mode of financing and evaluating researchers means that they are not inclined to verify research results. The certainty of scientific knowledge is not the effect of its corroboration, but of the process of rapidly closing and "stacking" black boxes. Instead of a critical approach, we are dealing here with a high level of mutual trust among the members of the research community. The dynamic of research fronts effectively raises the costs of any possible deconstruction or controversy.

All this does not, however, lead us to statements about the possibility of a cognitive closure of science—one more step is necessary. We have to remember about the ambiguity of results [cf. Fleck 1979; Lynch 1985; Knorr-Cetina 1981] and the high task uncertainty characteristic of many research fields, in particular those where knowledge is still being constituted and research methods are being institutionalized. Bearing this in mind, we obtain the model of a cognitive system that quickly turns the outcome of micro-negotiation occurring at the level of individual laboratories (to the degree they enter into the paradigm and do not stand in opposition to scientific assumptions) into a black box and stabilizes it through the effect of superimposition. In addition, we should remember the observations of Duhem and Hacking: Even if scientists are faced with an anomaly, a contradiction, or the necessity of revising an assumption, they have a range of techniques at their disposal for unifying fields of knowledge and avoiding cognitive dissonances.

An excellent exemplification of such processes is provided by studies of scientific controversies conducted by the Edinburgh School and the Bath School. Researchers rivaling one another in a controversy often refer to the same set of data, interpreting it to the advantage of the theory they are defending, or may function in two mutually incommensurate fields, working on the basis of different assumptions, knowledge, and instruments. Similarly, the choice itself of observations and the selection of test samples is directed by the hypotheses adopted. A situation often occurs where disputes can not be resolved on the basis of experimental results or observations: "Empirical fact" proves inconclusive. The closure of the controversy is achieved by such social

mechanisms as, among others, cutting off financial support, exclusion from the mainstream discourse, breaking off social ties, or even discrediting particular researchers [see, for instance: Barnes, Bloor, Henry 1996: 18–45; H. Collins 1983, 2000; Collins, Pinch 1998a; Latour 1983, 1988; Lynch 1985; Pickering 1981]. There is no room here to evoke the research of the Bath or Edinburgh schools. I therefore propose to focus on a single example that is a good illustration not only of the formation of a scientific black box, but also of cognitive closure and the autonomization of the scientific discourse from the empirical input.

A Case Study: the Scientific Discourse on the Cause of HIV/AIDS (1981–1986)[3]

The object of the case study is the scientific debate in the years 1981–1986 concerning the cause of AIDS and the process of converting the retrovirus hypothesis (the claim that the retrovirus[4] HIV causes AIDS) into a stable black box. At the beginning of the 1980s, when puzzling cases of illness among homosexuals began to be observed, explanations taking into consideration an infectious agent were considered unlikely. The syndrome was linked, above all, with the lifestyle of the risk group: The focus was on the intensive use of intravenous drugs, promiscuity, and the "fast" lifestyle among American homosexuals. It was only after a series of negotiations that it was possible to link the syndrome with a retrovirus. As we will see, legitimation of the retrovirus hypothesis occurred in the absence of, and independently of, empirical research. The discourse about AIDS constitutes an ideal example of the autopoietic communication system; it displays the self-production mechanisms of discourse in an almost pure form.

Two important issues should be emphasized here. First, the explanation of AIDS as an illness caused by HIV is an unusually strongly stabilized cognitive construct. It is reproduced and maintained, among other things, by medical practices that premise the detection and care of seropositive persons, ongoing medical research into HIV/AIDS and methods of preventing or combating it, references in the public discourse to the phenomenon, and widespread education on the subject of HIV/AIDS. The whole network of organizations that have formed around the assertion of a link between a specific retrovirus and AIDS should not be forgotten. Second, the acceptance and universal recognition of the relation between HIV and AIDS led to deep cultural, social, political, and even economic, transformations. Above all, sexual practices and mutual relations between partners underwent thorough changes. HIV/AIDS was re-

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[3] There is a large body of sociological literature on the scientific discourse on the cause of HIV/AIDS and the later scientific controversy around the dominant theory [see Altman 1986, 1994; Epstein 1995, 1996, 1997; Fujimura, Chou 1994; Murray, Payne 1988; Treichler 1988a, 1988b, 1991, 1992; Patton 1985, 1990; see also Collins 2000]. The article refers mainly to Steven Epstein's book *Impure Science* [Epstein 1996], which most completely describes the discourse and controversy around AIDS to the year 1995.

[4] Retroviruses are a family of viruses whose genetic material is contained in RNA. They conduct a process of reverse transcription: Thanks to enzymes called reverse transcriptase (or revertase) they transfer their genetic material from RNA to DNA, which then integrates into the genome of the host and with it undergoes replication.

peatedly used as a resource in ideological disputes: for instance, in the context of family values. The issues related to the epidemic were of unusually elevated significance in the context of the functioning of homosexual milieus and other risk groups and the manner in which they were perceived. It should be emphasized that as a result of social pressure to hasten drug approvals, the representatives of risk groups and infected persons were in a position to shape the very process of clinical trials in the USA, which had a far-reaching impact on the global pharmaceutical market [see Epstein 1995, 1996, 1997]. Finally, HIV/AIDS is unusually important in the context of the situation of third world countries; very often the problems of these areas are analyzed through the prism of the HIV/AIDS epidemic, and this affects the nature of actions taken.

If we consider the above two observations, it would appear that HIV/AIDS constitutes, at least from the sociological perspective, an extreme case. Here we have a situation where such an important scientific finding, whose result is unusually strongly maintained, was arrived at independently of empirical tests—it was the effect of the mechanisms of the discourse itself. Let us examine, therefore, the way in which not only the relation between HIV and AIDS was constituted, but also the epidemic itself.

From the "Los Angeles Five" to AIDS[5]

The Los Angeles Five and the Enigmatic Syndrome

From the SSK perspective, the beginning of the epidemic can be considered the announcement by the Centers for Disease Control and Prevention (CDC) that a new, enigmatic syndrome had been observed in five patients in Los Angeles [see *Morbidity and Mortality Weekly Report* no. 30 (5 June 1981): 250–252]. The patients were diagnosed with pneumocystosis (*Pneumocystis carinii pneumonia*, PCP), a pneumonia caused by an atypical fungus. Two died. The micro-organism causing PCP appears everywhere, but in normal conditions, the human immune system is capable of dealing with it. Two hypotheses were put forward. The first linked the puzzling cases of PCP with the fact that all the patients were active homosexuals. The second announced that the contagion could be connected with some sort of venereal disease. In alarm, the CDC personnel began to seek analogous cases of insufficient immunity or opportunistic infection.[6] Not long after, 26 further cases of mysterious illness were reported (20 in New York and 6 in California). The victims were young homosexuals suffering from Kaposi's sarcoma, a rare type of cancer usually diagnosed in the elderly. At least 4 of the patients had PCP at the same time, 8 died.

The public media began to report on the strange cases (among other papers, the *New York Times*, and the *New York Native*, a gay community periodical). They quoted the stance of the doctors, according to whom the epidemic could have something

[5] Reconstruction mainly after Epstein 1996.

[6] Opportunistic infection is an endogeneous infection characteristic of individuals with diminished immunity (in humans, it is chiefly caused by HIV). The pathogenic factors producing opportunistic infections (viruses, bacteria, fungi, protozoa) are most often not a threat to persons with normally functioning immune systems, but form a risk for persons whose immunity has been lowered. Such infections usually accompany cancers (sarcomas, lymphomas).

to do with an overload of the patients' immune systems through the accumulation of numerous venereal diseases, intravenous drugs use, or the popular "poppers"[7] [according to Epstein 1996: 46–47]. After succeeding reports, the epidemic gradually became a social fact that various social groups, institutions, and individuals had to take into account in their perception of the world. By the end of 1981 a whole series of articles had appeared in prestigious medical journals such as the *New England Journal of Medicine* (*NEJM*), which presented new information and speculations concerning the unknown illness. There was no doubt that a nationwide epidemic of immune deficiency was occurring. However, it was not known what caused the immunological impairment, how contagion was brought about, or what environmental factors came into play. Reports that the illness was not restricted to homosexuals started to flow in. 8% of the 159 cases recorded at the time were heterosexuals, including one woman. Reports also began to appear of atypical cases of PCP in persons using intravenous drugs (7 cases).

A new disease, or an epidemic itself, was not something that imposed itself on researchers—the cases of illness to that time were classified within the framework of existing epidemiological categories. From the SSK perspective, without the actions of the CDC and collaborating doctors, the epidemic would not so much have existed unnoticed but would not have constituted itself as a separate entity. That people fall ill and die is an obvious, "natural" social fact. However, what people fall ill and die of is the result of social negotiation. In this sense illness, medical conditions, contagions, and epidemics are performed by medicine. It is scientific and medical institutions that provide the content of various diseases' definitions [see, for example, Fleck 1979, Foucault 1994; Mol 2002; Mol, Law 1994, 2004].

From Immune Overload to the Virus Hypothesis

In spite of the above reports, attention was still focused on homosexuals and their fast-paced lifestyle. Homosexuality—it was reasoned—was as old as the world, while what was at hand was a new syndrome, and thus a new factor or factors must be at play. Increasingly often, hypotheses were formulated about an overload of the immune system. The syndrome was linked with "excesses" (drug abuse, the consumption of "poppers") and the "promiscuity" (viral infection, venereal diseases, and the strong medicines used to combat these conditions) generally attributed to sexually liberated homosexuals. It was conjectured that "life in the fast lane" destroyed the immune system and resulted in opportunistic infections. At the beginning of 1982, the disease was increasingly often being described by doctors as GRID—Gay-Related Immune Deficiency. In this way, the initial conceptual framework for explaining the cause and controlling the epidemic was formed.[8]

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[7] 'Poppers" is a term for various alkyl nitrates. The name was taken from the sound made from opening the hermetic packaging of these drugs. Among other purposes, they are inhaled to increase sexual pleasure. In the early 80s it was rather generally considered that they suppressed the organism's immunity.

[8] The main characteristic of a conceptual framework is that it organizes our experience and discourse. By definition, GRID was supposed to be an illness of homosexuals. Doctors had difficulty in acknowledging that the disease could appear outside a narrow risk group. Men who declared themselves heterosexuals

Homosexual communities quickly reacted to this disadvantageous definition of the situation by trying to question the connection between the syndrome and their lifestyle. The medical profession was accused of homophobia and hetero-sexism. As early as 1982, gays and lesbians began to organize foundations and associations whose aim was to help persons affected by the syndrome, but also, above all, to engage in political and media activities to shape the definition of the new illness and the homosexual image. Partially under the influence of homosexual communities and doctors who sympathized with them, the medical community rejected the politically incorrect name "GRID." It was replaced by the more neutral term AIDS, or Acquired Immunodeficiency Syndrome.

The transition from the paradigm of immune overload to the virus hypothesis occurred in 1982 and 1983. In 1983 there were reports of AIDS outside the United States. AIDS cases were discovered in 33 countries, on each of the inhabited continents. This information was published in all the prestigious medical journals, such as the *NEJM* and the *Lancet*. Among 29 cases noted in France, 6 patients had developed the syndrome in June 1981, that is, before the first news of the epidemic in the USA [see Epstein 1996: 55–56]. The information was a turning point. The previous conceptual framework organizing perceptions of the epidemic was repealed, making it possible to notice cases of the syndrome falling outside the original risk group. On July 9, 1983, the CDC announced 34 cases of Kaposi's sarcoma and other opportunistic infections among Haitians living in 5 different American states and among hemophiliacs who had intravenously received Factor VIII—a hematogeneous substance acquired by mixing the plasma of blood collected from a thousand donors.[9] According to some researchers, the transmission of AIDS was reminiscent of contagion by hepatitis B. Not only was this observation an argument in favor of the virus hypothesis, but it constituted the basis for formulating the first preventive measures (the use of disposable needles, limitation of the number of sexual contacts/partners) [Epstein 1996: 63]. The hypothesis of "one virus—one epidemic" shortly came to dominate.

This does not mean, however, that voices of skepticism did not appear. However quickly the virus hypothesis dominated the discourse, the immune overload hypothesis still had a group of adherents. The main actor here was Joseph Sonnabend. He argued that it was not very likely that one and the same illness should appear suddenly in four entirely different groups (Haitians, intravenous drug addicts, hemophiliacs, and homosexuals). He claimed that it could be a matter of four differing syndromes characterized only by similar symptoms [see Sonnabend, Witkin, Purtilo 1983]. Sonnabend, however, was not an epidemiologist. In the absence of credible research and data, his work was treated as scientific speculation. The remaining researchers defending the paradigm of immune overload began to be seen in a similar light. During the first

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but who were diagnosed with immunosuppression were suspected of hiding their true sexual preferences. There was resistance to broadening the extent of the epidemic to narcotics users with immunosuppression. Reports by pediatricians of observing immune deficiency in children were considered incredible.

[9] The CDC suggested that it might be a matter of the transmission of some infectious element in human blood. Since Factor VIII had been purified of bacteria, it was possible that there was a virus involved [Epstein 1996: 56–57].

two years of the epidemic not much research appeared that would support such an approach.

Retrovirology: LAV and the "Discovery" of HTLV-III

The factor causing AIDS turned out to be HTLV-III, a retrovirus whose discovery was originally ascribed to Robert Gallo of the National Cancer Institute (NCI).[10] Gallo's attention was directed to AIDS by James Curran of the CDC, who made the team from the NCI acquainted with the epidemiological reports and with his opinion that the cause of the syndrome was an infectious element. He pointed out that a characteristic of the syndrome was a lowered level of T lymphocytes. This inclined Gallo to take into consideration the hypothesis that the cause of AIDS was HTLV or another virus of that family. Gallo's assistant found the retrovirus in the T lymphocytes of several AIDS patients. Gallo reported his discovery in the pages of *Science* magazine. A succeeding article about traces of HTLV infection was written by his colleague, Myron Essex of the Harvard School of Public Health.

Tests of the lymphoid tissues of AIDS patients were also conducted by Luc Montagnier's team at the Pasteur Institute in Paris. The French team prepared T lymphocyte samples and bred the virus. Changes in the level of enzyme indicated that the virus had killed the cells and the presence of reverse transcriptase pointed to its being a retrovirus. By adding fresh cells, the French researchers were able to maintain the culture, and thanks to an electron microscope they could photograph the virus. Montagnier contacted Gallo to inform him of his discovery. Gallo encouraged the French virologist to report his discovery in *Science*. The articles by Gallo, Essex, and Montagnier were to appear simultaneously on May 20, 1983 [see Barré-Sinoussi et al. 1983; Essex et. al 1983; Gallo et al. 1983]. In reading them together, one could have the impression that the virus tested by Gallo was the same as that of which Montagnier was writing. However, the French scientists did not share this opinion. The tests had not confirmed that the "French virus" belonged to the HTLV family (the virus destroyed T lymphocytes, whereas HTLV caused them to multiply violently, leading to cancer). In September 1983, in Cold Spring Harbor, Montagnier announced that the virus called LAV (*lymphadenopathy-associated virus*), which had been noted by his team, was found in 60% of patients with lymphadenopathy (recognized to be the phase preceding AIDS) and 20% of patients with AIDS. None of the patients tested was infected with HTLV.

Gallo received from Montagnier a sample of LAV for testing. Shortly thereafter he announced the discovery of another virus in the HTLV family. He maintained that HTLV-III was the cause of AIDS. In January 1985, however, it was found that HTLV-III was characterized by a 99% genetic similarity to LAV. The viruses were too similar to come from different sources—obviously the virus discovered at the Pasteur Institute had found its way into Gallo's sample. It remains an open question

[10] Robert Gallo achieved renown as the co-discoverer of the retrovirus producing T-cell leukemia, a rare form of cancer. The virus was named Human T-Cell Leukemia Virus, HTLV for short. In 1982, he managed to find a succeeding virus of the same family, which—he maintained—produced another type of leukemia. The viruses were named HTLV-I and HTLV-II.

whether this occurred by accident (the adulteration of samples is a common problem in micro-biological laboratories) or whether Gallo was guilty of deception.[11] However, in 1984, this was not yet known. It is an unusually important point, because for many researchers the independent discovery of the same virus in samples by two different research centers would testify in favor of the retrovirus hypothesis. These two apparently independent scientific announcements mutually strengthened and legitimized one another. It was one of the factors that allowed the retrovirus hypothesis to acquire broad support in the scientific world, and the later revelation of the error could no longer damage the already partially closed black box.

The Heckler-Gallo Press Conference and Publication in Science

In 1984 another very important event took place: a press conference on April 23 organized by Margaret Heckler, then Secretary of Health and Human Services in the Reagan administration. Standing with Robert Gallo before a room full of reporters she announced that "The probable cause of AIDS has been found: a variant of a known human cancer virus, called HTLV-III" [according to Epstein 1996: 72]. (An analogous report appeared in the public press, including in the *New York Times*.) Heckler claimed that Gallo had managed to work out a method of reproducing the virus, as was necessary to develop a blood test for antibodies. In announcing the results of research publicly instead of publishing them in the pages of a reviewed scientific magazine, the Heckler-Gallo conference constituted a drastic violation of scientific etiquette. Publication occurred only on May 4, 1984 when as many as four articles by Gallo and his team appeared in *Science* [Gallo et al. 1984; Popovic et al. 1984; Sarngadharan et al. 1984; Schupbach et. al. 1984]. Taking advantage of the situation, Gallo put forward a series of hypotheses referring to the connection between HTLV-III and AIDS and possible tests for the presence of the virus in blood. In *Science* Gallo reported that he had managed to find and isolate the virus in the following cases: in 18 among 21 samples taken from patients in whom "lymphadenopathy syndrome" (recognized to be a phase preceding the real syndrome) had been found; in 3 among 4 mothers of juvenile patients with AIDS; in 3 of 8 juvenile patients with AIDS; in 13 among 43 adult patients with AIDS who had been confirmed as having Kaposi's sarcoma; and finally, in 10 among 21 adult patients with AIDS who had opportunistic infections. It might have been expected that HTLV-III would be present in each of the cases tested. Gallo considered, however, that most likely the test samples were not treated in the optimal manner for that particular virus. What is important is that clinically healthy persons were not found to have the virus.

According to the discourse at that time, this data did not yet prove that HTLV-III produces AIDS; it only showed the correlation between AIDS and the presence of the virus (or more precisely, its antibodies, as it was these the tests detected). It remained

[11] Finally, in 1985, the Pasteur Institute sued the US government. The suit concerned the patent rights for the test for retrovirus antibodies. In 1987, Jacques Chirac and Ronald Reagan signed an agreement dividing the royalties for the commercial antibodies test between their countries. In 1991, after it emerged that LAV had been in his test sample, Gallo was forced to make a gradual withdrawal of his claims to priority.

an open question whether the virus was the primary cause of AIDS or only another opportunistic infection—an innocent "free rider" virus. HTLV-III was theoretically supposed to destroy T lymphocytes in humans. It was not known whether it actually did so *in vivo*. The *in vitro* experiments with T lymphocytes and the virus seemed to confirm the theory, but the laboratory results were insufficient for the findings to be conclusive on the subject of the causal relationship.

Another researcher, Jay Levy, also sought the virus causing AIDS. In August 1984 he reported in *Science* [Levy et al. 1984] that he had managed to find traces of a retrovirus, which he called AIDS-associated retrovirus (ARV), in around half the AIDS patients he tested, in around 20% of clinically healthy homosexuals, and in scarcely 4% of healthy heterosexuals. Later, Levy's virus was pronounced identical with HTLV-III.

In 1986, in the face of such a large number of names and acronyms (LAV, HTLV-III, ARV, HTLV-III/LAV) the International Committee on Taxonomy of Viruses decided on a compromise name for the virus that—as it was then believed—caused AIDS. It was called HIV, i.e., Human Immunodeficiency Virus. This event constituted a certain stabilization point, or even an institutionalization of the retrovirus hypothesis. By that time, the syndrome, the virus hypothesis, and the epidemic itself had become stable elements of the social situation. The issues related to them were repeatedly raised in the pages of widely read newspapers and scientific journals. They were continually present in the news. They became a factor that politicians, medical practitioners, scientists and ordinary people had to consider, systematically, in their activities. At the same time, a whole new set of institutions focused on AIDS-related issues developed: specialist journals, research institutions, and grassroots initiatives of the gay community. In the years 1984–1986, the retrovirus hypothesis acquired universal acceptance in the scientific community, in the media, and in everyday discourse.

The Genesis of Proof, i.e., the Self-reference of Discourse Concerning HIV/AIDS

The Heckler-Gallo conference and the publication of four texts in *Science* by Gallo's team triggered sudden interest in the retrovirus. In the discourse, the significance of publications on the subject of the immune overload hypothesis declined. Meanwhile, the subject of the virus was increasingly often raised: In 1983, articles on the subject constituted only 2% of the entire press on AIDS while in 1986 they were 37% (nearly 2000 publications appeared). Simultaneously, from month to month, the retrovirus hypothesis became increasingly less problematical and in time achieved the status of a black box. Why, however, did the retrovirus hypothesis gain primacy?

The Truth-Will-Out Device: Did the Truth Come Out?

Did the retrovirus hypothesis begin to be generally accepted just because "it was true"? At the moment when knowledge is stabilized, scientists frequently make a reverse justification, explaining their own previous actions within the framework of the logic of scientific discoveries. At the moment a statement is stabilized, the final effect

of research or controversy is presented by scientists as an obvious, "natural" fact, which was imposed from the outset [see Fleck, 1979]. They thus erase knowledge concerning social negotiations, complicated research analyses, or the controversies around ambiguous results that usually accompany the emergence of new knowledge [Latour 1987]. A close analysis of the statements of scientists reveals that they have a tendency to mix together various disproportionate scientific explanations. Nigel Gilbert and Michael Mulkay [1982] showed that scientists, in explaining cognitive errors, the course of a controversy, and many other aspects of their work, make use of two basic rhetorical repertoires: the empiricist and the contingent. A scientist who uses the first repertoire presents his activities and convictions as entirely natural, suggesting that the obviousness of an empirical phenomenon will speak for itself [Gilbert, Mulkay 1982: 56]. The contingent repertoire, on the other hand, consists in presenting activities and convictions as being to a fundamental degree dependent on various non-scientific factors [Gilbert, Mulkay 1982: 57]. In criticizing scientific conceptions, the test subjects more often made use of the contingent repertoire, while in explaining their own position they made use of the empiricist technique. Cognitive errors—or at least, what was considered error—were most often explained using the contingent repertoire. The situation was similar in the case of scientific controversies. What is important, however, is that although both types of rhetoric would seem to be mutually exclusive, scientists most often used them in reference to the same problems. Often they were mixed together in one statement. This generated contradictions requiring mitigation. As a conversational analysis showed, when researchers switched from one type of explanation to another, a one or two-second break was sufficient to prevent them from noticing the dissonance. In analyzing the statements of the respondents, Gilbert and Mulkay also caught more sophisticated techniques for mitigating or avoiding conflict. One of the patterns of the discourse analyzed was called the "truth will out device" (TWOD) [see Gilbert, Mulkay 1982: 109–11; see also Sojak 2004: 227]. The scientists stated that in the research situations they described a large role was played by outside considerations (social, cultural, emotional, or organizational factors). They expressed the conviction, however, that time was in favor of substantive factors. In other words, in spite of their perception of social and cultural determinants of scientific knowledge, they considered that in the final account these do not influence the shape of conceptions and research findings. They claimed that time would show which views were true. This is confirmed by the observations of other SSK representatives. For example, Latour [1987] described research practice similarly. He distinguished science in its ready-made form from science in action. Established knowledge is most often explained by scientists themselves by means taken from the empiricist repertoire. Scientists more often draw from the contingent repertoire in the case of knowledge that is only just being constituted. Furthermore, during a controversy, researchers are more often described in categories of rationally calculating, profit-oriented, culture-determined or emotion-driven actors than as disinterested cognitive entities guided by the logic of scientific discovery. It was no different in the case of the HIV/AIDS hypothesis. A good example is Gallo himself. In his opinion, there was already enough data in the early years of the epidemic to support the virus

explanation. Similar opinions were expressed by other discourse participants. At the same time, the earlier approach was treated in categories of cognitive error, which could have been avoided by adherence to scientific method. In other words, the dominant approach was explained in the framework of the empiricist repertoire. On the other hand, the contingent repertoire was used in reference to the rejected etiologies of AIDS.

From the SSK perspective it is hard to consider such explanations satisfactory. The principle of symmetry [Bloor 1991: 7] assumes that part of a conviction can not be explained by referring to an objective state of nature, while the rest is explained in categories of errors, omissions, or social interests. Because a piece of knowledge is considered obvious does not explain why it came to be so considered. SSK studies therefore often ignore or even challenge science's auto-presentation. Would it be possible to formulate an explanation of the success of the retrovirus paradigm without reproducing explanations based on the TWOD and reverse rationalization?

The Dense Network of Mutual Citations and Positive Modalization of the Retrovirus Hypothesis

Paula Treichler formulated the following hypothesis: A small group of scientists, constantly quoting each other's work, quickly produced a dense network of reciprocal citations and thus achieved very early control over the scientific discourse on AIDS and its etiology—its nomenclature, publications on the subject, and invitations to conferences [see Treichler 1992: 76]. Treading the path pointed out by Treichler, the subsequent statement can be risked: Through mutual citation of one another's work, a group of scientists created a social sense that the retrovirus hypothesis they defended was unproblematic; an external observer could come to the conclusion that since there was such a rich literature on the subject, the basic premise must be justified. This legitimizing, retrovirus-hypothesis system based on a thickening network of internal references among a closed circle of researchers was so effective that it did not require additional support in the form of new scientific reports. Thanks to this, a statement that was originally regarded by its authors as comparatively unlikely and requiring further research gradually acquired the status of objective truth without additional proofs.

Steven Epstein conducted a thorough analysis of the discourse around the cause of AIDS. He analyzed the content of articles that appeared in seven of the leading scientific journals in the years 1984–1986 and referred to Gallo's article on the causal relation between HIV and AIDS published on May 4, 1984 in *Science*. In sum, Epstein analyzed 244 articles, including 16 from *Nature* and 66 from *Science*. His study concentrated on the way in which Gallo's retrovirus hypothesis was referenced. Over half (57.6%) of the texts he studied from 1984 referred to the statement that a retrovirus was the cause of AIDS, but only 3.4% of the texts openly referred to the statement without reservations. In 1985, 24.5% of texts already referred to Gallo's retrovirus hypothesis openly and without reservations. Over half the texts (58.5%), however, still referred to it reservedly. In 1986, scarcely one fifth of the texts (21.5%) retained any reservations. As much as 62% of the analyzed works openly and unquestioningly accepted Gallo's thesis [Epstein 1996: 79–104].

Manifestations of skepticism or alternative explanations of AIDS were unusually rare in the test sample.[12] In practice, researchers undermining the dominant perspective had to expect sanctions from the scientific community [see the case of Shyh-Ching Lo; Epstein 1996: 92]. In sum, the authors referring to the May 1984 text in *Science* addressed the thesis of Gallo, Montagnier, or Levy with a greater or lesser degree of acceptance. As the discourse developed, the retrovirus hypothesis was unconditionally accepted with increasing frequency.

It is important that succeeding articles gradually reinterpreted the meaning of the earlier reports and analyses. More precisely, the later articles ascribed stronger statements concerning the causal relationship to the earlier publications than the authors of the cited texts had been in a position to make. For example, in 1985, *Science* contained a text written by several epidemiologists from the CDC, who stated in the introduction that the retrovirus named HTLV-III, known also as ARV and LAV, had been isolated a year earlier and shown to be the cause of AIDS. A similar group of researchers from the Virology Department of the Food and Drug Administration wrote in 1985 that the role of HTLV-III in the development of AIDS had been firmly established. In the opinion of the authors of both texts, Montagnier, Gallo, and Levy had already proved it in their texts [according to Epstein 1996: 83]. However, at this stage, the claim of a relationship between HIV and AIDS constituted only the most likely thesis, not a proven and recognized scientific fact. None of the texts the authors referenced (or any of the 244 articles analyzed by Epstein) gave conclusive proof of the HIV/AIDS hypothesis.[13] Furthermore, the cited authors themselves did not claim that they had formulated anything that could be taken as proof of a causal relationship between HIV and AIDS.

We are dealing here with what Latour described as processes of adding, deleting or changing modalities. Scientists address the claims of their fellow researchers with various types of modalities, both positive—"it is true that x," "x has important theoretical consequence"—and negative—"it is doubtful whether x," "it is impossible that x," "it seems to the authors that x." Positive modalities shift the claim away from the conditions of its formation and present it as an objective fact, removing all traces

[12] Only one article, published in 1984, considered the possibility that the virus hypothesis could be erroneous [Epstein 1996: 80–83]. In the *Lancet*, the Heckler-Gallo conference was criticized as a media spectacle; a call was made for a more careful and sober appraisal of the available data. It was emphasized that discovery of the virus in selected samples did not yet constitute proof of a causal relationship. Finally, however, the *Lancet* editors, taking into account the fact that two laboratories had independently isolated the same virus, were inclined to assume that it was this factor that was responsible for the epidemic (it was not then known that Gallo's samples had been contaminated with LAV). A skeptical article and letters also appeared in *NEJM* and in the *Journal of the American Medical Association* (*JAMA*). In March 1985, Luc Montagnier wrote carefully in *JAMA* on the subject of the pathogenesis of AIDS. A little later he stated that some additional cofactor could participate in the development of AIDS: for instance, antigens or foreign proteins. In other words, Montagnier tried to supplement his retrovirus hypothesis with certain elements of the immune overload hypothesis [according to Epstein 1996: 84].

[13] In writing about the lack of conclusive proofs or the paradoxes of the HIV/AIDS paradigm, I am not referring to any external, "objective" standards of scientific research or philosophical conceptions on the methodology of the natural sciences. I am not evaluating whether something can be considered a proof or not, but only reconstructing, after Steven Epstein, the manner in which participants in the given scientific field treated their own research and how they qualified it.

that it constitutes someone's creation. Negative modalities evoke the conditions in which a given thesis was created—with the result that it appears methodologically problematic or less objective [see Latour 1987: 23]. An extremely positive or negative technique of modalization consists in passing over a given claim in silence. In the case of an extremely negative reception, the thesis is greeted with a silence that means it is so absurd as to be beneath notice. The most obvious facts are also not mentioned; they are treated as manifest claims on which successive theses can be superimposed. The process of superimposition means that the facts of the claim become an increasingly stable black box. In the discourse analyzed by Epstein, we have such a situation: The retrovirus was positively modalized in various ways by the participants in the discourse. It was gradually transformed from a likely hypothesis into a proven fact. For many, it became so obvious that it was not worth discussion.

During the time period under analysis, not much new information or research appeared that could be recognized, on the grounds of the premises and standards accepted in the said discourse, as evidence proving or giving plausibility to the initial hypothesis of Gallo, Levy, and Montagnier. Simultaneously, researchers were still struggling with unresolved problems: (1) it had not been possible to isolate the virus in half the patients who had developed the syndrome, (2) theoretically, the virus was supposed to produce the syndrome when the T lymphocytes were attacked, but by using the most sensitive of available methods, the researchers had been able to find the virus only in an unusually small number of cells taken from a patient (sometimes in scarcely one lymphocyte in 100,000), (3) the virus destroyed lymphocytes *in vitro*, but in the case of living organisms, the effect was not as obvious—too small a number of cells turned out to be infected, and (4) serological researchers were not able to show that it was an invasion of the virus that produced immunosuppression [Epstein 1996: 88–89]. In addition, the HIV/AIDS hypothesis did not fulfill Koch's postulates.[14] These postulates are a stable element of the medical tradition and are used to exclude the possibility of blaming a disease on the wrong factor, for instance, a passenger virus. The HIV/AIDS hypothesis did not meet the first and third postulate (the virus was found in scarcely half the patients, and it was not possible to create an animal model) [Epstein 1996: 88–90].

In spite of the above, the process of positive modalization of the retrovirus hypothesis continued. In the years 1984–1986 all the problems and "paradoxes" of the retrovirus hypothesis were viewed not as insurmountable barriers but as ordinary anomalies. No other hypothesis was as well established or offered the prospect of rapid containment of the epidemic. The HIV/AIDS paradigm held the hope that tests, antiviral medications, and even a vaccine against HIV could be delivered in a matter of years or months. In addition, the original works were layered over with articles citing them. In effect, the HIV/AIDS relation began gradually to be seen not

[14] According to Koch's postulates, for a given infectious element to be recognized as the cause of a disease, (1) it should be found in every case of infection, (2) it should be isolated and bred in the form of a pure culture, (3) the culture thus prepared should infect a susceptible laboratory animal, which should then develop the disease, and (4) the infectious element must be isolated from the infected animal [see Walker, LaVine, Jucker 2006].

as a probable hypothesis but as an obvious premise constituting the starting point for further research. The discourse shaped in this way produced the deep feeling that the causal relationship must have been empirically well grounded. In practice, however, no work yet existed that could be recognized as a confirmation of the hypothesis.

A similar situation existed in regards to the immune overload hypothesis; in that case too no new research appeared to support the hypothesis. The difference consisted in the fact that the overload hypothesis was rejected while the retrovirus hypothesis was maintained, even though it did not generate, over a comparable time period, conclusive scientific proofs. The lack of proof is an argument against a hypothesis only and exclusively when a given research milieu has the will to raise the question.

The Self-legitimization of the Discourse About AIDS

On the basis of Epstein's analysis, the hypothesis could be risked that the biomedical discourse on the cause of AIDS functioned as a self-referential system. Its point of reference was not so much an external reality (represented thanks to experimental procedures and other research techniques) but its own elements, i.e., publications and other scientific statements. The discourse was not anchored in the world under study—rather, the discursive system constituted the basis for defining an image of the world and the medical definition of the syndrome.

Scientists, instead of referring to new research, chiefly cited the earlier—already classical—research of Montagnier, Gallo, and Levy, or one another. For example, much of Gallo's later work was conducted chiefly to systematize the previous research; it did not bring any qualitatively new data to the discourse [according to Epstein 1996: 86–87]. In this manner there arose, on a foundation of a few texts considered classical, a dense and cohesive network of authors and texts basing themselves on the practice of mutual citation. The system formed the basic legitimization of the thesis placed in the discursive framework. In practice, individual texts referred to other texts as those that constituted conclusive proof. Thus there arose a continuum of references and circular citing [cf. Epstein 1996: 87]. The authors referred to each other in a criss-crossing manner as the ones who had provided final proof. On the other hand, a situation was achieved where the first text was cited by others who treated it as a scientific proof, while the text itself referred to other, more original, but not more conclusive, work.

The previous observations can be summarized in the following manner: The main supports of the discourse were the classic, founding texts of Gallo, Levy, and Montagnier. A network of mutual references was established on these works. However, the process of positive modalization and imputation meant that the texts were viewed not only as formulations but simultaneously as confirmations of the retrovirus hypothesis.

What is interesting here is the role that Gallo played in this discourse. A considerable part of the literature analyzed by Epstein (a third of the texts that appeared in 1984 and 1985 and a quarter of the texts in 1986) were works by Gallo, or his colleagues, with whom he had earlier published on the subject of the retrovirus hypothesis. Gallo's name or those of his colleagues were often to be found somewhere in the middle or end of a long list of co-authors. (In many cases, Gallo's role may have been limited to the provision of research samples.) However, the channels for

spreading Gallo's idea in the medical and scientific community through personal ties or face-to-face contacts are obvious here. Due to this network of contacts, Gallo's group could strengthen the status of the retrovirus hypothesis in the scientific discourse. At the same time, they could increase their credibility simply by publishing an enormous quantity of material and continually referring to their own work.

Neither the deluge of literature on the subject of the retrovirus hypothesis nor the process of shifting the modality are anything unusual and do not constitute a scientific pathology. Similarly, in other areas, scientists seek to stabilize their hypotheses through a flood of publications on a given subject. During the period when a large literature on a subject is being created, a given phenomenon begins to be treated by researchers as an objective element of their practice [see Latour 1987, 1999]. However, scientists are striving above all to persuade other researchers to build research on their claims in order to stabilize them additionally. Where there is a high degree of consensus around a hypothesis in spite of the lack of proofs, a process can occur like this one, which increased the costs of questioning the retrovirus hypothesis.

A "Self-confirming Hypothesis" or the Facilitation of Scientific Proof

In the years 1984–1986 there were not yet proofs (or what could pass for proofs on the basis of the prevailing methodological standards) of the retrovirus hypothesis—it was confirmed only over the course of the following decade. Let us observe, however, that there is an enormous difference between producing scientific proof in favorable social conditions, when nearly the whole community (not only the scientific community) expects confirmation of a given hypothesis, and attempts to legitimize the claim in a situation where there is a lack of consensus or in conditions of fierce controversy. In the first case, it is much easier to gain the interest of fellow researchers, favorable reviews, or funds for research; in the second case, scientific opponents will constantly subject the results of research and experiments to criticism, hampering both reference to the results of experiments and observation, and the support of fellow researchers. Even if it is possible for a research team to confirm a hypothesis satisfactorily, other teams could just ignore the results, or prove incapable of replicating them in their own laboratories (remember that the replication of innovative research requires time to master the new technique and acceptance of the possibility that the result we are trying to repeat is not simply an artifact).

Referring to Robert K. Merton [1948] we can state that we are dealing here with a certain variety of social phenomenon known as the self-fulfilling prophecy. Application of this concept to the process of closing a scientific controversy could seem counter-intuitive. Let us elaborate this issue. As was shown above, the publication of the papers by Gallo and his team in *Science* triggered processes that resulted in the formation of the discourse based on a network of mutual positive citations. This in turn created a widespread sense (inside and outside the scientific community), that one was dealing with an established fact. The general sense of the truth of the retrovirus hypothesis not only encouraged attempts to prove the hypothesis, but above all facilitated their realization. Simultaneously, the situation hindered the activities of possible opponents, who had difficulties accessing research resources and were

exposed to social sanctions. Let us return, however, to the category coined by Merton. A self-fulfilling prophecy is a social situation where the spread of a conviction concerning the state of the world causes people to begin to behave in such a manner that in effect the existence of this state becomes more probable, even if the original conviction was unfounded. The classic example is the spread of a prediction/rumor that a given bank will go bankrupt in the near future. However, as Thomas C. Schelling observed, in certain aspects, a better term for the phenomenon would be that of "self-realizing expectations" [Schelling 1991: 38]. It is also more useful from the perspective of the present analysis. It should be remembered that according to Merton's conceptualization, the claim about the state of the world triggering the change is originally erroneous and becomes true only through the collective actions of people. In the case of an analysis of a scientific claim's black boxing, one can not step outside the discourse and state whether the thesis was true or not—there is no external instance to which one could refer. What interests us is the relation between the expectations of the scientists and the process of stabilizing a given hypothesis as an objective, indisputable fact. I would also like to emphasize that I am not claiming that faith in the accuracy of a hypothesis led automatically to its legitimization. It is not only the number of citations, but also the results of experiments and scientific observations that influence the status of the scientific claim [see the "blood flow" model, Latour 1999: 98–108]. I am only stating that to a large degree a general consensus and societal expectations increase the probability of proofs being provided. This claim does not imply, however, that the discovery of proofs linking HIV to AIDS was only a matter of time. Rhetoric of "the truth will out" type is fairly widespread among scientists. SSK and STS make us look at the causes of closure not in the structure of unknown reality, as yet unarticulated by scientific instruments, measurements, or experiments, but in the actions of scientists themselves and other social groups. We can only ponder the question: If the course of the debate on the cause of AIDS had been slightly different, would it be the hypothesis of immune overload that is universally accepted today?

Could the Case of AIDS Discourse be Described in Categories of Objectivist Epistemology?

The question remains: Could the above situation not be equally well explained using categories taken from the objectivist model of knowledge [see Zybertowicz 1995: 58–71]? In attempting such an interpretation several problems would have to be dealt with. The first is that a cognitive consensus on the HIV/AIDS relationship was formed in the absence of binding scientific proof. Of course, it could be argued that it was in the end shown that HIV causes AIDS: The theory is today much better grounded than in the period under analysis. But how could scientists have propounded such a result in the years 1982–1986? The second problem is how to explain the success of the retrovirus paradigm in the perspective of the defeat of the immune overload approach? Both approaches turned out, in the analyzed time period, to be degenerating research programs [cf. Lakatos 1977]. New proofs on their behalf did not appear and—more importantly—they did not make it possible to deal with the epidemic. Both paradigms formulated certain partially converging techniques

of dealing with the problems of the epidemic. It can be supposed, however, that an appraisal of their effectiveness was done within the framework of the self-legitimizing discourse.

From the position of the objectivist model of knowledge the following accusation can be leveled at this argument: Could what is being suggested by any chance be that the epidemic and the relation between HIV and AIDS was created, or even "made up" by scientists? A certain explanation is necessary here. The present analysis is not dealing with the world as understood in objectivist categories. It does not concentrate on the "world in itself" or "the reality lying underneath," but on certain of its models generated by the research community. As was shown by Bruno Latour [1987], from the SSK and STS perspective, reality is not a resource for settling controversies, but rather it is an outcome of scientific negotiation—by closing black boxes scientists define what will generally be taken as reality. The present text is not a work in the field of epidemiology, virology, or the methodology of the natural sciences. I am not trying here to answer the question of whether there is a relation between HIV and AIDS or whether it is only an artifact or hoax. It was likewise not my intention to evaluate the scientific competence of representatives of the given field of research. The real object of analysis was the procedures and patterns of discourse that allowed the objectification of the retrovirus hypothesis. In writing about objectification, I do not mean that something was physically shaped, constructed or created. The term "objectification" refers exclusively to the status that was generally ascribed to a given object within the framework of the discourse under analysis.

Conclusion: the Cognitive Closure of Science

In the present essay, we have reconstructed some of the social factors that produce a situation where the findings of individual laboratories can be quickly transformed into black boxes. Contrary to the general image of scientific culture and the criticism that has been linked to it, at least since Karl Popper, in the case of many fields of research there exists a systemic encouragement to build black boxes instead of opening discussion or trying to disassemble what is held to be a hermetically sealed black box. The manner in which scientists will approach research is partially derived from the economics of the "science game" characteristic of their research field. As STS has shown, there is no such thing as a universal set of procedural rules for all scientific fields. Research fields differ in terms of their own organizational ecology. As Stephan Fuchs has shown, this "ecology" consists in the nature and availability of research resources, as well as the probability of success (or what may be considered success on the grounds of the principles of a given discipline). In addition, the variations in what could be called the research style (in accordance with Ludwik Fleck), the manner in which a given field is represented in the public sphere, and what ties it has with such areas as industry, should be taken into account.

The example of the controversy over the cause of AIDS shows that research fields often function on the principle of self-legitimization and self-reference, which leads

to their cognitive closure to the world. As we have seen, this discourse was configured in such a way that researchers, through mutual reference and theoretical imputations, caused the basic premise about the cause and effect relationship between HIV and AIDS to begin to be universally accepted as an indisputable fact. Two issues are key in this context. First, the retrovirus hypothesis was accepted in spite of a lack of conclusive proofs—at least according to the officially declared methodological standards of the given field. In other words, through the effect of "stacking" black boxes and shifting modalities, the HIV/AIDS hypothesis became an unrevisable fact on which a series of scientific findings and social institutions were constructed. Second, faith in the hypothesis facilitated, in the long term, its final verification. Attention should be paid to the fact that in the case of the debate around AIDS an important role was played by public representation. We are not dealing here with a situation where scientists publicly announced results only after obtaining certainty; hypotheses, which were contested by part of the community, were presented as theses around which a broad consensus existed. The problem is not only the premature announcement of the results, but also the pressure that was exerted on the researchers. In such conditions the process of closing a black box acquires an entirely different dynamic and scientific assumptions are even more quickly made into unchangeable facts.

The case of the debate around the cause of AIDS is in certain respects exceptional. It exemplifies the functioning of an autopoietic cognitive system in the domain of scientific research in an almost pure form. Is it, however, an isolated instance? Analyses could presumably be made of the ways in which a consensus was formed around many other scientific theses having far-reaching political, cultural, or economic consequences. It is worth quoting here Robert Gallo's answer in 1988 to researchers questioning HIV as the direct cause of AIDS: "As anybody in the business knows who works [with AIDS], there is more evidence that this virus causes AIDS than you have with the majority of diseases [that we] long ago have accepted [as caused by viruses]" [according to Epstein 1996: 90]. Of course, the answer quoted here is a rhetorical measure aimed at representing the HIV/AIDS relationship as so obvious as not to be worth discussing. However, if Gallo's statement is treated as representative, it could be asked how many other theses concerning the mechanisms by which disease is produced were converted into black boxes in an analogous manner. Furthermore, we do not need to limit ourselves exclusively to an analysis of biological epidemics. The controversy around the etiology and even the status of ADHD, as well as many other psychiatric assumptions [Kirk, Kutchins 1992], could be indicated here [see Afeltowicz, Wróblewski 2013].

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