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The burden of proof and the origin of acquired immune deficiency syndrome

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There is a distinct difference in the way that different theories about the origin of acquired immune deficiency syndrome have been treated, with the widely supported cut-hunter theory given relatively little scrutiny, while the oral polio vaccine theory has been subject to intense criticism. This difference in treatment cannot be explained as application of the scientific method. A better explanation is that the burden of proof is put on all contenders to the cut-hunter theory, giving it an unfair advantage, especially given that this assignment of the burden of proof appears to reflect non-scientific factors.

Keywords: origin of AIDS; burden of proof; polio vaccines; scientific proof

1. ORIGIN OF ACQUIRED IMMUNE DEFICIENCY SYNDROME STORIES

Since the first cases of acquired immune deficiency syndrome (AIDS) were diagnosed in 1981, the disease has killed millions of people, so it is natural to ask how it originated. Aside from intellectual curiosity, understanding of the origin may help in developing means of curing or preventing AIDS or in preventing the outbreak of similar diseases.

The most commonly supported origin story is the transfer of simian immunodeficiency viruses (SIVs) from non-human primates to humans, where they become human immunodeficiency viruses (HIVs), by any of several methods, such as a hunter butchering a monkey and getting blood from it into a cut, a monkey biting a human or a person eating undercooked monkey meat. This transfer is then thought to have led to the AIDS pandemic through human-to-human transfer. This will be called here the cut-hunter theory—a metonym for direct non-human-primate-to-human transfer followed by human-to-human amplification—since the more usual expression 'natural transfer' suffers from semantic confusion due to multiple meanings of 'natural'; furthermore, human social processes are integral to the theory.

There have been numerous competing theories for the origin of AIDS, including, for example, that a biological warfare experiment went wrong and that factors other than HIV are responsible for AIDS (Lederer 1987, 1988). For the purpose of analysing the scientific reception to competing theories, the focus here is on one particular alternative: that mass vaccination campaigns in Africa in the late 1950s, using an oral polio vaccine (OPV), were responsible for the AIDS pandemic (Cribb 1996; Curtis 1992; Elswood & Stricker 1994; Goldberg & Stricker 2000; Hooper 2000; Lecatsas & Alexander 1989; Pascal 1991).

The argument for the OPV theory goes like this: OPVs were cultured on monkey kidneys and thus could have been contaminated by SIVs; monkeys with SIVs do not necessarily show any sign of ill health and so may not have been rejected as sources of kidney tissue; the vaccine was not tested for SIVs, which were not discovered until 1985; OPVs were given to around one million people in Central and western Africa in the period 1957–1960; the earliest known AIDS cases and HIV-positive blood samples are strikingly correlated with the time and location of the African immunization campaigns; many young children, whose immune systems are undeveloped, were given the vaccine in an extra high dose. In addition, vaccine transmission of monkey viruses to humans is known to be possible because OPVs contaminated by the monkey virus SV40 were given to millions of people (Shah & Nathanson 1976). This theory first received widespread attention in 1992 and was given renewed attention on publication of Edward Hooper's book *The river* (Hooper 2000).

In a number of publications (e.g. Basilico *et al*. 1992; Hahn *et al*. 2000; Koprowski 1992; Korber *et al*. 2000), the OPV theory has come under close and critical scrutiny. However, there appears to be no equivalent examination of the cut-hunter theory. The aim of this paper is to explore this difference in treatment. A brief overview of ideas about methods and rhetoric in scientific proof is given in § 2, § 3 analyses the burden of proof in two characteristic publications, and the concluding section (§ 4) spells out some implications.

2. METHODS AND RHETORIC IN SCIENTIFIC PROOF

Over the past several decades, historians, philosophers and sociologists have learned a lot about the process of doing science, with insights that often conflict with the perceptions of practitioners and the general public (Barnes 1974; Chalmers 1976; Collins & Pinch 1998; Hess 1997; Ravetz 1971). Scientists often explain what they do as an exercise in applying the 'scientific method', but many scholars who have examined the practice of scientific research conclude that the method is more rhetoric

than reality (Barnes 1974; Bauer 1992; Feyerabend 1975). Scientific research is a complex activity that cannot readily be boiled down to a set of rules; practices vary depending on the discipline, the research topic and often the scientist. Formal rules can account for only a component of scientific behaviour, since much that occurs is unspoken and indeed unconceptualized (Polanyi 1966).

In much research, scientists seek evidence to confirm their hunches, yet philosophers have long demonstrated that confirmations do not prove a theory, since there may exist evidence that conflicts with it. An opposite approach, championed by Karl Popper (1963), is to seek evidence that conflicts with hypotheses. Popperian falsificationism is often invoked by scientists to explain what they are doing, even when it is not an accurate reflection of what they are actually doing (Gilbert & Mulkay 1982; Mulkay & Gilbert 1982). One of the problems with falsificationism is that there is no definitive evidence. Conflicting evidence can be explained away as faulty experimentation or interpretation, like dismissal of the careful 1930s experiments that showed ether drift, apparently falsifying special relativity. If the conflicting evidence is hard to dismiss, a theory can be maintained by introducing subsidiary hypotheses, rather like Ptolemaic epicycles used to prop up the theory that the Sun revolves around the Earth. Then there is the case of uniformitarian geology and evolutionary biology in the second half of the 19th century. Leading physicist Lord Kelvin calculated the age of the Earth to be far less than geologists and biologists had been assuming, leading most of them to adapt their theories to Kelvin's shorter figure. The later discovery of radioactivity led to a vast increase in the accepted age of the Earth (Burchfield 1975).

Not only can evidence be discounted, explained away or superseded by new discoveries, but it is never independent of theory (Hesse 1974). A fact can only be understood through the lens of a theoretical framework. This means that competing frameworks may interpret facts in different ways. The classic account of competing frameworks is Kuhn's (1970) study of paradigms, normal science and revolutions in science. While the concept of paradigms has been debated and modified (e.g. Barnes 1982), the idea that facts are theory-laden is standard in social studies of science.

Another factor affecting scientific research is the intense commitment of scientists, especially leading scientists, to their ideas (Mahoney 1976; Mitroff 1974; Watson 1938). Scientists are likely to defend their pet theories against criticisms and new evidence, finding ways of preserving their preferred options in the face of tremendous challenges. This can actually be functional for science (Mitroff 1974), in that promising options are not discarded too easily but rather kept alive by their die-hard adherents. There are examples, such as continental drift, where a few lone partisans held out against orthodoxy and whose ideas later became orthodoxy.

Given that facts are in part dependent on theories, that unwelcome facts can be challenged or dismissed, that theories can be maintained despite disconfirming evidence, and that many scientists are highly committed to their preferred theories, there is an abundance of evidential, conceptual and psychological resources for waging scientific disputes, not to mention material resources including laboratories, salaries and publishers. Scientists can 'push' their arguments by their choice of technical assumptions, through selective use of evidence and results, by their way of referring to alternative arguments and through their treatment of uncertainties (Martin 1979). The implication is that choices between scientific theories are not 'scientific' in the sense of being purely logical choices made by neutral researchers based on unambiguous evidence and clear criteria. While it may be a goal to move towards such a model, in practice things are much messier epistemologically and pragmatically.

One of the advantages of better understanding the dynamics of scientific knowledge creation is the possibility of throwing light on ongoing scientific disputes. Rather than operating with an idealized picture of scientific practice, the complexities and biases can be acknowledged and used to help achieve the goals of science, which, arguably, should include both better knowledge and benefit to humans (Horrobin 1990; Maxwell 1984).

3. THE BURDEN OF PROOF IN ORIGIN OF AIDS THEORIES

When two or more theories are in competition, it is common for one of them to be treated as the established position—the default option, as it were—and the others to be treated as challengers. A challenging theory is normally expected to bear the burden or onus of proof. In other words, advocates of the challenging theory are expected to provide highly convincing evidence and arguments before the theory can be taken seriously. To use a different metaphor, it is assumed that the established theory has jumped over a very high hurdle to gain its leading position and that any challenger must jump over an equally high hurdle before being in contention for the remainder of the race.

While this sounds reasonable, the problem in practice is that scientific criteria alone are inadequate for deciding whether a theory should or should not bear the burden of proof. In many cases, a theory becomes the established position through chance or social factors, such as evidence for it being discovered earlier or its advocates having easier access to publication. Furthermore, when two theories are in competition, there are no unambiguous criteria for deciding when the burden of proof should switch from one to the other. Hence, making an assumption about the burden of proof can be a means by which scientists 'push' their arguments and thereby promote their favoured theory (Martin 1979).

These abstract considerations have an immediate and direct application to theories about the origin of AIDS. Nearly all commentators have assumed that cut-hunter transfer is the default option, with the burden of proof carried by any challenger. Yet the cut-hunter theory never went through any significant scrutiny in order to gain its leading position: no 'hard evidence' has ever been produced to show a hunter was exposed to SIV leading to transmissible AIDS. It might be said that advocates of the cut-hunter theory sneaked under the initial high hurdle but are demanding that all other contenders jump over it. Whatever the process, assumptions about the burden of proof have a major impact on comparative assessment of

scientific theories. To illustrate this, two publications are examined here: the Wistar Committee report (Basilico *et al*. 1992) and a recent paper by Hahn *et al*. (2000).

(a) *The Wistar Committee report*

The Wistar Institute in Philadelphia produced the polio vaccines that, according to the OPV theory, may have been responsible for triggering the AIDS pandemic. Following publication of an article about the theory by Curtis (1992) and subsequent publicity, the Wistar Institute set up a committee—the AIDS/Poliovirus Advisory Committee—to examine the theory. The Committee produced a short report in September that year (Basilico *et al*. 1992). That this report is based on the assumption that the OPV theory bears the onus of proof can be illustrated by three features.

First, the report examines the OPV theory but does not examine the cut-hunter theory. If the burden of proof had been equally shared, then equal critical attention should have been devoted to the cut-hunter theory.

Second, the report concludes that the OPV theory is 'extremely unlikely' to be correct by using an *a priori* analysis of probabilities, but does not apply this method to the cut-hunter theory.

The authors seek to determine the probability that the OPV theory is correct by 'assessing the probability that each step in this postulated mode of transmission would have occurred successfully to allow HIV or a close progenitor to enter the human population during the Congo poliovirus vaccine trials' (p. 1). The principal steps they consider are contamination of the vaccine by SIV, transmission of SIV and HIV by the oral route, and mutation of any known monkey SIV to HIV-1 in the time between the vaccination campaigns and the earliest samples of HIV-1. They assess the probability of each step as low and hence the probability of the concatenation of steps as extremely low. This approach is internally logical but the conclusion drawn about the probability of the OPV theory requires an additional, unstated assumption, namely that the probability of cut-hunter transfer is significantly higher.

If the same approach of assessing the probabilities of steps in a chain of transmission is applied to cut-hunter transfer, then some principal steps to consider are that the transfer occurred in the precise geographical region from which the AIDS pandemic appears to have originated, that the transfer occurred just before the earliest known HIV-positive samples and cases of AIDS, and that an additional transfer occurred (for HIV-2) at roughly the same time. If, for the sake of argument, it is assumed that monkeys with SIVs have been butchered for 100 000 years, then the chance that SIV would cause a pandemic precisely in the past century is, *a priori*, 1 in 1000 (setting aside considerations such as rates of butchering and riskiness of butchering techniques). The chance that two independent SIV transfers to humans causing a pandemic or epidemic would occur within the same century then becomes 1 in 10^6. This figure does not yet include the factor of geographical distribution nor additional independent SIV transfers. (A more realistic period for human–primate predation is one or two million years, but the presence of SIVs over this period is speculative.)

Of course, such a calculation applied to the cut-hunter theory is unfair, because we know now that AIDS first occurred in the past century. An *a priori* probability calculation is not sensible, since it is possible to work backwards and say that the SIV transmissions must have happened just at the times that would lead to the present manifestations of disease. However, if this sort of *post hoc* analysis is applied to cut-hunter transfer, then it should also be applied to competing theories. If two routes of transmission each have a 1 in 10^6 chance of occurring, neither is likely. But if these are the only two possible routes and it is known that transmission did occur, then logically they should be considered to be equally likely.

The Wistar Committee thus dismisses the OPV theory by loading it with an enormous burden of proof, namely the requirement that it be probable *a priori*, while applying no such burden to cut-hunter transfer, implicitly assumed to be the default theory.

The third feature is that the Committee uses a single contrary piece of evidence as a definitive refutation of the OPV theory. The Committee stated: 'The most telling evidence is the case of the Manchester sailor who appears to have been infected with HIV-1 even before the poliovirus trials were begun in Congo' (p. 6). The sailor, David Carr, died in 1959 of AIDS-like symptoms first displayed in 1958. His tissues were later found to contain HIV (Corbitt *et al*. 1990).

As noted in § 2, any theory can be rescued in the face of contrary evidence by rejecting or dismissing the evidence or by introducing subsidiary hypotheses, namely by suitably modifying the theory. In 1992, there were several ways that the evidence of the Manchester sailor could have been explained away while still maintaining the OPV theory. The earliest mass uses of OPV vaccine in Africa were in 1957, and David Carr could have had sex with a vaccinee during his naval career and rapidly developed AIDS. Alternatively, he might have been given a contaminated polio vaccine in one of the early experimental trials in Britain. Finally, the HIV detected in his tissues might have been a contamination.

That the detection of HIV in David Carr's tissues was later refuted (Zhu & Ho 1995) is not the main point here since, even without that finding, the OPV theory could have been maintained using any of various explanations for the finding, or by just ignoring the evidence for the time being, as is common practice in many scientific disputes. The key point here is that the Wistar Committee did not make any attempt to explain away the Manchester sailor evidence, something that can be interpreted as reflecting its assignment of the burden of proof to the OPV theory.

In summary, the entire argument of the Wistar Committee report is shaped by its authors' assumption that the OPV theory bears the onus of proof. There is a striking asymmetry in the way that competing theories are examined: only one theory is scrutinized; the theory is subjected to an *a priori* probability analysis that is extremely difficult to overcome; and no attempt is made to find ways around a 'telling' piece of evidence. If the OPV theory had been the established one, and the same sort of approach applied to its challengers, the cut-hunter theory could have been dismissed equally easily.

(b) *Hahn* et al. (*2000*)

Hahn *et al*.'s (2000) paper 'AIDS as a zoonosis' is an analysis of transmissions of SIVs to humans, with discussions of the genetic characterization of SIVs, the origins of HIVs and implications for science and public health. It assesses two AIDS-origin hypotheses: cut-hunter transfer and OPV. The onus of proof is put on the OPV theory, as shown by the following three points.

First, Hahn *et al*. expect 'direct evidence' to support the OPV theory but not the cut-hunter theory. They say that the OPV theory relies 'on the supposition that chimpanzee and sooty mangabey kidneys were used in vaccine preparation, although there is no direct evidence to support this contention' (p. 612). They do not mention any of the circumstantial evidence for use of chimpanzee kidneys presented by Hooper (2000). On the other hand, they do not present any direct evidence for cut-hunter transfer nor even say whether it would even be possible to obtain direct evidence. Thus the OPV theory is held to a higher standard than the cut-hunter theory.

Second, they use an estimate of the date of HIV-1's origin apparently calculated assuming cut-hunter transfer as an argument against the OPV theory. They say 'the M group of HIV-1 has been estimated to have originated 10–50 years before the OPV vaccine trials were conducted' (p. 612). This dating apparently assumes a single initial simian–human transfer, consistent with cut-hunter transfer. However, OPV transfer could have involved dozens or hundreds of near-simultaneous transfers, significantly changing the dating of the origin of the M group. Hahn *et al*. put the burden of proof on the OPV theory by expecting it to meet challenges established assuming the correctness of the cut-hunter theory.

Third, they use arguments from a single discipline to reject the OPV theory, which is a multidisciplinary option, but do not reject the cut-hunter theory using arguments from a discipline other than their own. They argue that the subtypes of HIV-1 group M are unlikely to have resulted from different chimpanzee SIVs injected into humans via polio vaccines, due to the genetic equidistance of the observed subtypes. Setting aside a technical response to this argument, it can be noted that it assumes rejection of the OPV theory is possible simply through genetic arguments. The extensive epidemiological evidence for the OPV theory (Hooper 2000) is not addressed. In contrast, Hahn *et al*. support the cut-hunter theory with detailed genetic arguments but a superficial treatment of historical and social factors that must be invoked to explain why, if AIDS originated early in the 1900s, it did not spread more widely much sooner than observed. Furthermore, they do not address the arguments drawn from African history that suggest the implausibility of AIDS being restricted to a few villages for decades before spreading more widely (Cribb 1996; Hooper 2000). This asymmetrical treatment of the two theories can be explained by Hahn *et al*.'s assumption that the burden of proof lies with the OPV theory. This means that the OPV theory must meet every objection from every discipline—in particular Hahn *et al*.'s own genetic arguments—whereas the cut-hunter theory need not meet objections from disciplines outside the authors' fields of expertise.

Like the Wistar Committee report, Hahn *et al*.'s (2000) arguments are shaped by their assumption that the OPV theory bears the burden of proof. This leads to a striking asymmetry in treatment of the two competing theories. 'Direct evidence' is expected of the OPV theory but not of the cut-hunter theory; a calculation of the date of origin apparently relies on the assumption of cut-hunter transfer; and arguments from a single discipline (genetics) are used to reject the OPV theory but arguments from a single discipline (social history) are not used to rebut the cut-hunter theory. If the positions of the two theories were reversed, it would be equally easy to reject cut-hunter transfer.

4. CONCLUSION

The OPV theory for the origin of AIDS has been subject to close critical scrutiny and, it should be said, this is quite appropriate in science. On the other hand, the competing cut-hunter theory has not been subjected to a similar scrutiny, but rather treated as the default option. Relatively few attempts seem to have been made to confirm cut-hunter transfer empirically, for example by finding pre-1950s' HIV-positive blood samples, nor to falsify it. This can be explained by proposing that scientists supportive of the cut-hunter theory have been successful in placing the burden of proof on challengers. Interestingly, though, there has been little urgency in seeking to confirm or falsify the OPV theory by testing samples of early polio vaccines. Calls to test samples held by the Wistar Institute were first made in the early 1990s (Curtis 1992), but apparently were not heeded until 2000.

Since the OPV theory was proposed, several developments have occurred in its favour, including refutation of the evidence that the Manchester sailor had AIDS (Zhu & Ho 1995), the uncovering of suggestive evidence about the use of chimpanzee kidneys to make polio vaccines (Hooper 2000) and collection of epidemiological evidence concerning the earliest known cases of AIDS and HIV-positive blood samples (Hooper 2000). No equivalent developments have bolstered the case for the cut-hunter theory, for which direct evidence seems virtually impossible to obtain. Therefore, it can be argued, it would be appropriate to reverse the onus of proof or at least to subject the cut-hunter theory to scrutiny equivalent to that given to the OPV theory.

The question arises, why has the OPV theory been expected to carry such an enormous and continuing burden of proof? One answer is that the theory is quite threatening to members of the scientific and medical establishment because it stigmatizes medicine for causing AIDS and reduces public trust in vaccinations (Martin 1993, 1998). Evidence compatible with this explanation for the treatment of the theory includes threats of legal action and actions against authors and publishers of the theory (Curtis 1995), repeated rejection of submissions (Martin 1993, 1998) and statements by critics that the theory would be detrimental to current vaccination efforts (e.g. Hooper 2000, pp. 436, 783; Vaughan 2000, p. 240) or cause distrust in science (Moore 1999). Proponents of the theory have presented various reasons why it should be treated more seriously, including gaining insights for opposing AIDS and alerting people to the

dangers of new iatrogenic diseases, for example through xenotransplantation.

Like many other scientific controversies, the debate over the origin of AIDS contains a mixture of scientific and social assumptions and argumentation. Trying to separate the scientific and the social may seem attractive but has the danger that social factors may simply be buried in what seem to be scientific matters, of which the burden of proof is a distinctive example. An alternative is to be more open about all the assumptions being made, and to accept a wide range of interested parties, both scientists and non-scientists, as legitimate participants in the debate.

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