

laboratory currently reporting tritium in the high range found at Texas A&M is the Bhabha Center in India. The rest of the "many" labs have either reported small incremental levels of tritium or have formally reported nothing at all. Scientists can only judge the reliability of data through publication of these data in peer-reviewed scientific journals. What is needed is the reporting of data and experiments that can speak for themselves, and a year and a half after the "discovery" of cold fusion those data and experiments are still talked about but not seen.

As for the work of E. K. Storms and C. Talcott, referred to by Bockris and sent to me as well, these researchers spiked a heavy water electrolysis cell with tritiated water, while monitoring the tritium concentration as a function of time. As expected, what they observed is a constant baseline level before the spiking, an instantaneous rise in tritium concentration (associated with the spiking), and then a linear decay in concentration after spiking, due to the dilution of the tritium by periodic addition of heavy water to replace that lost from electrolysis.

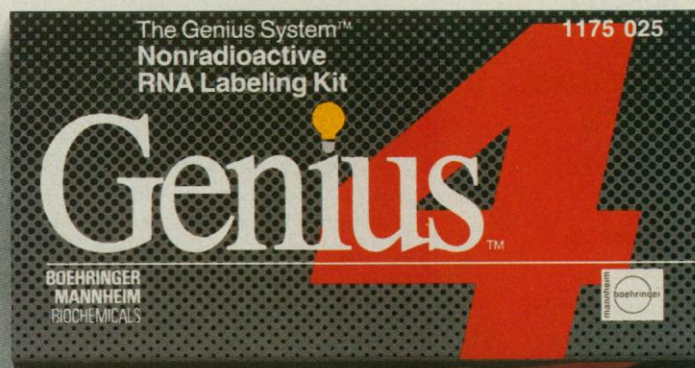
Storms, Talcott, and Bockris argue that Bockris's tritium data first show a rapid decrease in tritium content, and then a linear decrease due to the dilution by subsequent addition of heavy water. They interpret this rapid decrease as the release of deuterium-tritium gas generated in the cell, and the presence of this deuterium-tritium gas as incompatible with the spiking model.

This argument, however, does not appear to be substantiated by the experimental data. Bockris has shown only a single cell for which enough data points exist to define the shape of the tritium decay curve. In that cell, only a single point exists above the linear decay line. It is not scientifically justifiable to use a single data point to claim the existence of an effect, in this case a second decay lifetime. Therefore, using that second decay lifetime, the existence of which has not been established, to further prove that the tritium cannot be due to spiking is even less justifiable. —GARY TAUBES

#### **Viral Etiology of AIDS and the Gallo Probe**

The News reports by Barbara J. Culliton (22 June, p. 1494) and Ellis Rubinstein (22 June, p. 1499) contained illuminating, new information about the evolution of our knowledge about the viral etiology of AIDS. As an old virologist who has worked on human viral diseases for almost 60 years, I believe that the issue is not the honesty and

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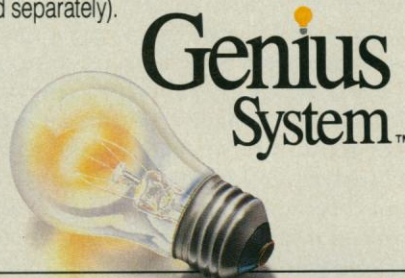
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integrity of Robert Gallo, who has been my friend for 20 years. In my view, the issues are (i) Who discovered the human retrovirus, now known as HIV-1, that was subsequently established as the usual cause of AIDS? (ii) What special "reagent" made it possible to establish this new virus as the etiologic agent of AIDS by classical virologic procedures, who discovered this reagent, and who first used it for this purpose?

The answer to the first question is unquestionably Luc Montagnier and his associates at the Pasteur Institute in Paris. They correctly identified the transverse transcriptase-producing agent they isolated from an AIDS patient (LAV/BRU) in normal human lymphocyte cultures as a human retrovirus distinct from HTLV-I and HTLV-II. However, the normal human T lymphocyte cultures were a poor medium for primary isolation and for subsequent propagation of the virus in sufficient concentration to produce antigen for serologic tests. Accordingly Montagnier and his colleagues, in their first publication in *Science* (1), correctly stated that "the role of this virus in the etiology of AIDS remains to be determined."

Thus, knowing what technology made it possible to establish the French LAV/BRU

virus, and others related to it structurally and antigenically that were isolated subsequently, as the etiologic agents of AIDS is crucial. The answer is that it was the use of an *existing*, established and continuously growing human CD4<sup>+</sup> lymphocyte cell line, in which the cells were not destroyed by the virus as in normal human lymphocyte cultures and in which these new AIDS viruses multiplied in high concentration. According to Rubinstein, Dean Mann of the National Cancer Institute (NCI) advised Mika Popovic in Gallo's laboratory to use such a cell line in October 1983; Jay Levy of the University of California in San Francisco *independently* used such a cell line (HUT78) for isolation of AIDS virus in November 1983 (2). The HUT78 lymphocyte cell line used by Levy was developed by Adi Gazdar, Desmond Carney, and Paul Bunn and was maintained in continuous culture by Gazdar at NCI, who deposited it in the American Type Culture Collection (ATCC) in March 1982. However, the HUT78 cell line that was in the Gallo laboratory freezer in 1981-1983 was, according to Mann, contaminated with other cells. The growth of the French LAV strain of the virus in Gallo's apparently contaminated HUT78 cell line by Betsy Read, a Gallo technician, in October-November, 1983 was said to be only "transient" and inadequate. Instead of obtaining the uncontaminated cell line from the ATCC, Popovic spent much time trying to obtain a suitable, uncontaminated cell line by terminal dilution technology and finally came up with a cell line that he called the H9 "clone." According to a special National Institutes of Health (NIH) committee chaired by Alan Rabson of NCI, the H9 cell line was identical with HUT78. This is not a trivial or irrelevant matter, as it was called by Gallo and Popovic, because the use of such uncontaminated, continuous lines of human T4 lymphocytes was crucial to the regular isolation of the new retrovirus from patients with AIDS and to the development of the antibody tests by which it was possible to establish the *etiological association* of the virus discovered by Montagnier and his colleagues and of subsequently isolated viruses related to AIDS. Once this technology had become available for the regular isolation of this new group of viruses from AIDS patients and for the demonstration of the regular development of antibodies, the rest of the work could be termed "kitchen virology." Popovic and Gallo, Levy, virologists at the Center for Disease Control in Atlanta, and Montagnier and his colleagues all contributed to this important effort, regardless of whether the data were published a few months earlier or later.

The question of whether the virus used by

Popovic and Gallo for the antibody procedure patented by the NIH was one virus or a hybrid of several viruses recovered by Popovic from a mixture of viruses during his search for a strain that would yield the highest concentration of antigen, or whether that mixture (*unknown* to Popovic and Gallo) also contained the French LAV strain as a contaminant, is of more commercial than scientific relevance. In any case, the molecular biological evidence summarized by Culliton and Rubinstein leads me to conclude that the "balance of probability" is that the so-called American "Popovic-Gallo" strain is the same as, or is largely derived from, the French "Montagnier LAV" strain of the AIDS virus.

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#### REFERENCES

1. F. Barré-Sinoussi *et al.*, *Science* **220**, 868 (1984).
2. J. Levy *et al.*, *ibid.* **225**, 840 (1984).

If one defines opportunism as a capacity to capitalize, or to capitalize on serendipitous discoveries, Robert Gallo might be criticized for having done so extremely well with the HUT78 strains of neoplastic lymphocytes. Similarly, *Chicago Tribune* journalist John Crewdson might be criticized for having capitalized on Gallo's difficulties. However, Crewdson's investigations and Rubinstein's article of 22 June have brought to light some of the difficulties that scientists face in communicating with one another, especially when large groups are involved and fame or fortune is at stake. One can only hope that the magnitude of the present AIDS and adult T cell leukemia pandemics will improve mutual communication for the benefit of all concerned.

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*Erratum:* In the chronology that accompanied the news report on the origin of the cell line in which Robert Gallo's laboratory first grew the AIDS virus (Ellis Rubinstein, "The untold story of HUT78," 22 June, p. 1499), Doris Morgan and Frank Ruscetti are incorrectly credited with the characterization of interleukin-2. In fact, they were the first researchers to show that you could grow T cells long term. But it was Dartmouth researcher Kendall Smith and his team who subsequently figured out what the active ingredient was in the conditioned medium—an ingredient that, in a 1978 paper [*J. Immunol.* **120**, 2027 (1978)], they called T cell growth factor. The molecule that acts as a T cell growth factor was named interleukin-2 by a consensus of attendees at a lymphokine meeting held in Switzerland in 1979.

*Erratum:* In Robert Holt's review of B. R. Grant and P. R. Grant's *Evolutionary Dynamics of a Natural Population* (20 July, p. 300), the second paragraph should have begun, "Baldly stated..."

*Erratum:* Beth A. Snyder, not Ralph L. Nicholson, should have received credit for the cover photograph of 29 June 1990.