

# Rethinking AIDS

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**Does HIV exist? Do HIV tests indicate HIV infections?  
Here's why some scientists say no.**

## ***THE ISOLATION QUESTION***

**How an Australian biophysicist and her simple observations  
have taken center stage among AIDS reappraisers**

by Paul Philpott

*Of course HIV exists—I've seen pictures of it in text books and on the news—and scientists work with it every day. How could there be HIV tests if there's no HIV? What those tests detect, that's HIV...*

So goes the typical response from physicians, biologists, and AIDS activists when faced with a very simple question: Does HIV exist? But like all questions fundamental to the HIV/AIDS model, nobody asked this in 1984, the year Robert Gallo published a group of four papers in *Science* (224:497-508, May 4) proclaiming the existence of a unique retrovirus, HIV, that causes AIDS.

Gallo's HIV-AIDS model stood unquestioned in the medical literature for three years, until 1987, when UC-Berkeley retrovirologist Peter Duesberg published the first academic paper contesting the notion of pathogenic retroviruses (*Cancer Research* 47: 1199-1220). Although disputing the infectious AIDS model, Duesberg accepted Gallo's claim of having prepared *isolates* of a unique retrovirus, HIV, and having abstracted from them proteins needed to construct tests for identifying people and cells infected with it.

By 1987 the plasma and T4 cells of thousands of AIDS patients had been tested for evidence of the proteins and genetic material from Gallo's "isolates." The AIDS reappraisal movement grew out of Duesberg's critique of these data. HIV exists, but the

blood contains so little of it, and it infects so few T4 cells, and replicates—harmlessly—*in vitro* with so much difficulty, and so many patients test negative for it altogether, that it is just too ineffectual, inactive, and imperfectly correlated with AIDS to explain AIDS.

### **Out of Australia: Questioning HIV's existence**

Before Duesberg's 1987 paper made it to press, a second academic, authoritative deconstruction of HIV had already been submitted for publication in

another journal. This one was written by Eleni Papadopulos-Eleopulos, a medical physicist at Australia's Royal Perth Hospital. In 1988 France's *Medical Hypotheses* (25:151-162) published her paper, "Reappraisal of AIDS: Is the Oxidation Induced by the Risk Factors the Primary Cause?" Papadopulos had independently reached many of Duesberg's conclusions, but ultimately had quite a different take on Gallo's claims: "Unlike other viruses [HIV] has never been isolated as an independent stable particle."

What she meant was this: Electron microscope pictures, *micrographs*, of samples Gallo calls "HIV isolates"—and of all "HIV isolates" produced before by Luc Montagnier of France, or since by other scientists—show some objects that look like retroviruses (the "HIV") plus lots of

## **PAPADOPULOS DIGESTED**

### **ABOUT THE AUSTRALIANS**

Eleni Papadopulos-Eleopulos, MSc, is a biophysicist and professor in the Department of Medical Physics at Royal Perth Hospital, a teaching hospital at the University of Western Australia.

Valendar F. Turner, MD, was until recently a practicing emergency room physician at RPH and professor of emergency medicine at UWA. Today he is affiliated as a consultant emergency physician.

John M. Papadimitriou, PhD, MD, is a practicing pathologist and professor at UWA's medical school.

David Causer, PhD, is Senior Physicist, Head of Medical Physics, and professor at RPH. Both he and Papadimitriou are experts on electron microscopy.

Together they have published many scientific papers since 1988 refuting the infectious AIDS model and questioning the existence of HIV. They argue that AIDS and "HIV" both result from tissue oxidation caused by such things as recreational and pharmaceutical drugs (including "anti-HIV" drugs like AZT), hemophilia clotting factor, transfused blood, rectally deposited semen, repeatedly acquired infections, and malnutrition. They propose anti-oxidant therapy as a treatment for patients diagnosed with AIDS conditions.

other things, including things that clearly aren't viruses. So there's no way to identify the origin of the "HIV" proteins and genetic material abstracted from these samples. Do the proteins come from the objects that look like retroviruses? Or do they represent some of the contaminants?

And what about those retroviral-looking objects? Papadopulos pointed out that among the microbial objects that look like retroviruses are (1) microvesicles: non-infectious, unstable organelles that bud from cells; and (2) *endogenous* retroviruses: non-infectious, unstable retroviruses coded for by healthy human DNA. She noted that this presents a special problem for the objects called "HIV." They can be observed only in cell cultures that have been stimulated by agents that induce the production of microvesicles and endogenous retroviruses.

Without true isolates of the objects declared "HIV," there really is no way to determine if they constitute what HIV is claimed to be: a retrovirus of *exogenous* origin (an autonomous entity unaccounted for by a person's inherent DNA library). There is no way to pull proteins and genetic material out of a heterogeneous sample and know that they came from one group of particular looking objects rather than another, or simply from the surrounding molecular soup.

### Oxidative stress:

#### Unifying AIDS, its causes, and "HIV"

In addition to introducing an HIV critique based on the principal of viral isolation, Papadopulos also unveiled in her 1988 paper an explanation for AIDS based on the process of oxidative stress. According to Papadopulos, the stimulants used to induce "HIV" phenomena (retrovirus-looking objects plus

## Isolating a virus

To isolate a virus, scientists take a heterogeneous sample (fluid from a patient or a culture) and add it to a graduated density gel, which they spin in a centrifuge. The contents of the sample settle into separate piles, or *bands*, at different depths according to their characteristic densities. These bands are called *density-purified samples*.

Because all microbiological entities have characteristic densities, scientists can obtain density-purified samples that contain only certain viruses, and no other material. There is only one way to confirm this: a photograph with an electron microscope that contains *nothing* but identical virus-looking objects.

If the micrograph reveals contaminating entities, that means the sample contained some material that had the same density as the virus-looking objects. In that case, scientists would have to add additional steps to the isolation process, ones that purify based on other characteristics—like size, or electrical affinity—until they could produce a sample that contained only the virus-looking objects. However, this usually is not necessary. Density purification typically produces true isolates of virus-looking objects.

If the density, appearance, and size of these objects match those of a previously characterized virus, scientists can label the sample a *virus isolate*. If not, scientists must subject the sample (actually, a fresh sample, since electron microscopy destroys whatever it photographs) to a battery of tests to prove that the virus-looking objects are viruses.

certain proteins that may or may not be affiliated with those objects) in cultures are *oxidizing agents*. As are the factors uniting American AIDS patients, including street drugs, hemophilia treatments, and rectally deposited semen. Papadopulos proposed that both "HIV" phenomena and AIDS conditions are consequences of these and other stressors she would introduce in later papers (such as blood transfusions, anti-AIDS pharmaceuticals including AZT, and antibiotics).

Duesberg drew on the 1988 Papadopulos paper (and even earlier writings by John Lauritsen in the gay press) in formulating his 1992 treatise "AIDS Acquired by Drugs and Other Non-contagious Risk Factors" (*Pharmacology & Therapeutics* 55:201-277). In that paper, Duesberg added to his HIV critique alternative explanations for AIDS. He agreed with Papadopulos that street drugs and hemophilia treatments caused AIDS, but dismissed rectal insemination as inconsequential. His 1992 paper was the first to implicate "anti-HIV" drugs such as AZT, and Papadopulos subsequently adopted them into her oxidative stress model.

That same year, 1992, Papadopulos formed a writing team with two University of Western Australia physician-professors, Valendar Turner of the Department of Emergency Medicine, and John Papadimitriou, Professor of Pathology. Together they published "Oxidative Stress, HIV, and AIDS" (*Res-Immunol.* 143:145-148), which restated her Unified AIDS Theory.

### Proving an isolate consists of viruses

Looking like a virus is just one feature of a virus. To be a virus, virus-looking objects must behave like a virus, and their constituents must relate to each other in special ways. Scientists demonstrate these criteria by adding an isolate of virus-looking objects to a culture of suitable cells. If the isolate consists of viruses, they will infect the cells and multiply to numbers much greater than those present in the original isolate.

Scientists confirm this by attempting to re-isolate the virus-looking objects from the culture after enough time has passed for substantial viral replication to have taken place. The new isolate should form at the same density as the original, and contain objects that look the same as those in the original sample. But the new isolate should consist of a much thicker band, indicating a larger number of viruses.

Scientists also have to examine the constituent molecules of the isolate. Among other things, they have to confirm that the DNA or RNA codes for all the proteins,

This being the case, scientists declare that the objects are indeed viruses, and that these viruses are characterized by a certain size, shape, and appearance, and consisting of a particular number of proteins and genetic molecules of certain molecular weights or base pair lengths.

## Virus tests without virus isolation?

In 1993 Papadopoulos finally caught the attention of AIDS reappraisers. "Is A Positive Western Blot Proof of HIV Infection?" appeared in *Bio/Technology* (11:696-707), a major medical journal and sister publication of *Nature*.

The article debunked the validity of "HIV tests" on several grounds: (1) that they are constructed from the constituents of heterogeneous samples rather than true viral isolates; (2) that proponents of the purported virus (HIV) claim to observe it only in stimulated cultures, as opposed to fresh patient plasma; (3) that accuracies for these tests are established without an independent *gold standard* (isolation from fresh patient plasma); and (4) that these tests are assumed to be equally accurate for people with and without the risks associated with, and the conditions classified as, "AIDS," a syndrome the purported virus supposedly causes.

Isolation, Papadopoulos explains, is the only sure proof that a virus is present—the only direct, unambiguous evidence of a virus. And isolation from *uncultured* patient plasma is the only sure proof that a person harbors an active infection—the *only* sort of infection that can cause disease. She points out that the accuracy for even a properly constructed viral test (one made from true viral isolates) can be established only by answering the following question: In what fraction of people who test positive can the virus be isolated from their fresh (uncultured) plasma?

Instead, "HIV" test accuracies are established using circular logic; "accuracy" for HIV ELISAs is taken as the fraction of positive people who subsequently test HIV Western blot positive. And "accuracy" for HIV Western blot tests is nothing more than reproducibility (the fraction of positive people who test positive when retested).

These pseudo accuracies—each over 99%—are assumed for

## Proving a virus causes a disease

If a virus is hardy and abundant enough to cause a disease, scientists should have no trouble isolating it from the cell-free fluids of affected tissue. This is exactly what scientists must do to convict a virus of causing a disease. They select a group of people who have the disease, and try isolating the virus from the patients' *plasma* (cell-free blood), or other fluids, depending on the disease. If they fail to isolate the virus from some of the patients, then they must absolve that virus of responsibility for any progressive disease in those individuals, and conclude those people are sick for some other reason.

But what about patients who *do* present isolatable amounts of the virus? Is that virus responsible for their conditions? Or is the virus an innocent bystander? After all, most viruses cause no disease.

Only microbiological experimentation can establish the culpability or innocence of a virus isolated from the fluid of diseased tissue. To do this, scientists prepare cultures of healthy, uninfected cells of the type damaged or destroyed in the patient. They add viral isolates and watch to see if this effects the culture cells in ways that can explain the disease.

## Isolating viral constituents

To isolate the contents of a virus, scientists must dismantle the viruses into their constituent molecular parts. They do this by adding a special detergent, SDS, to a viral isolate. The isolate will then consist of the individual molecules that compose the viruses. These molecules include proteins that decorate and line the outer membrane envelope, the globs that form the hollow inner core, and the contents of the inner core: enzymes and DNA or RNA.

Next scientists separate these molecular species from each other, using *electrophoresis*, whereby an electric field pulls the molecules through a gel so that they band according to their *weights* (instead of densities). Some of the bands will contain proteins, and others contain genetic material, either RNA or DNA.

Scientists call an electrophoresed sample a *Western blot* if they are considering the bands that contain proteins, a *Southern blot* if they are considering the bands that contain DNA, and a *Northern blot* if they are considering the bands that contain RNA. (The unusual names are a salute to E. M. Southern, the scientist who devised this process.)

Protein bands and their constituent molecules are named according to the weight (in daltons) of the molecules. The prefix "p" stands for *protein*, and "gp" stands for *glyco-protein* (*glyco* meaning that the protein has some sugar molecules stuck to it). RNA and DNA bands are named according to the number of nucleic acids or base pairs (in kilobases) that make up the constituent RNA or DNA molecules.

all people, even those free of the risks and symptoms associated with the syndrome that the purported virus supposedly causes. Yet among risk group members with blood that reacts with these tests—those who test positive—pseudo isolations ("HIV" phenomena in stimulated cultures) are achieved for only some of those with AIDS conditions, and for only a few who are symptom-free.

For example, of risk group members (gay men, drug injectors, and blood recipients) testing "HIV-positive":

(1) Gallo achieved pseudo "HIV" isolations in 26 of approximately 63 (41%) patients with AIDS conditions (this is a generous figure that assumes Gallo's isolations involved only the 88% of his 72 AIDS-diagnosed patients who tested positive);

(2) Piatak reported (a) "infectious HIV" (according to some of the same criteria as pseudo isolations) in only 29 of 38 (76%) patients with AIDS conditions and in only two of 21 (10%) patients with no AIDS conditions (*Science* 259: 1749-1754, 1993); and (b) in one of six (16%) symptom-free patients (*Lancet* 341: 1099, 1993);

(3) Daar reported "infectious HIV" in none of four symptom-free patients (*NEJM* 324[14]:961-964, 1991);

(4) Clark reported "infectious HIV" in none of three symptom-free patients (*NEJM* 324[14]:954-960,

1991); and

(5) Cooper found "infectious HIV" in neither of two symptom-free patients (*Lancet* 340:1257-1258, 1992).

So among people with AIDS risks, using pseudo isolations from stimulated cultures as an independent standard, HIV antibody tests are between 41% and 76% accurate for people with AIDS conditions, and between 0% and 16% accurate for those with no symptoms, a far cry from the 99% accuracies established using reproducibility and cross-checking.

What about people without AIDS risks? No one has compiled even pseudo isolation data for drug-free, blood product injection-free heterosexuals who test positive. HIV researchers simply assume that the data from risk group studies apply for everyone.

And what about the *real* accuracy of HIV tests? That is, accuracy established using the only valid gold standard: isolation from fresh plasma. The Australians reason that since isolation from fresh plasma has not been achieved under any circumstance, then the true accuracy for *all* "HIV tests" should be considered *zero*, and all positive results should be regarded as false. There is no basis for thinking that a virus observed only in stimulated cultures exists in the plasma of any humans, even those who test positive for it as determined by antibody, antigen, "viral load" or any other assay.

### "HIV": Normal cellular residents?

In the *Bio/Technology* paper, Papadopoulos examined what are accepted as substitutes for true HIV isolation. These include "HIV proteins" (gp160, gp120, gp41, p32, p24, and p17), reverse transcriptase, "HIV" DNA and RNA, and retrovirus-looking objects. She suggests that they are each cellular constituents, some normal, some produced in response oxidative stress.

(1) HIV existentialists—those who think HIV exists—hypothesize that gp160 is made of gp120 stuck to gp41, and it decorates HIV, with gp41 embedded in the outer membrane envelope, anchoring gp120, which protrudes outward, ready to latch onto T4 molecules; Papadopoulos cites references showing that gp160 and gp120 are oligomers of gp41 (four gp41s stuck together make gp160; three make gp120), and that gp41 might be the ordinary cellular protein actin. (She also cites references showing that cell-free objects considered to be HIV contain no gp120, and thus have no infectious capability, just like endogenous retroviruses.)

(2) The existentialists hypothesize that p17 lines the inside of the envelope, and p24 forms the hollow core; Papadopoulos cites references showing that p24 and p17 might be the two constituent globs that form the ordinary cellular protein myosin.

(3) The existentialists hypothesize that p32 decorates HIV's en-

## Understanding viral tests

Although isolation is the only direct evidence of a virus, cost and time considerations make it impractical for clinicians. Among other things, for example, it requires confirmation by an electron microscope.

Viral tests, on the other hand, are much simpler. Most require clinicians to just add patient fluids (usually plasma, depending on the virus in question) to the tests and look for reactions to take place.

Scientists construct these tests using components abstracted from viral isolates. Some of the proteins from viral isolates, for example, will react with antibodies secreted into plasma by the immune systems of patients infected by the virus. Antibody tests consist of those proteins. Genetic tests consist of probes made from the DNA or RNA contained in viral isolates. The probes react with viral RNA or DNA in patient fluids.

When constructed and validated properly, and used under the proper circumstances, viral tests can be nearly as accurate and reliable as viral isolation itself. The need for proper test validation and result interpretation stems from the fact that the reactions upon which they depend (antibody-antigen interactions, and genetic probing) are not perfectly specific. Antibodies against one viral protein can react with a similar protein from other microbes, or even some non-microbial proteins. Similar proteins mean similar gene sequences, so genetic tests are less than perfectly specific as well.

Furthermore, even when tests react with their intended viral entities, this doesn't necessarily mean the patient has an active infection, the only sort that can cause disease. Antibodies, for example, can circulate for years—even a lifetime—after the host immune system has suppressed a viral infection to permanent and harmless latency, or even eliminated it entirely. Viral genetic material can also persist in the plasma and other fluids during viral latency.

Therefore, viral tests cannot absolutely and unambiguously identify an actively infected person. Only isolation of objects that possess the appearance and density of the virus in question can do that. So viral tests must not only be constructed from viral isolates, they must also be validated against their ability to predict patients from whom scientists can obtain viral isolates.

Validation studies tend to show that positive test results are highly accurate for patients who express the symptoms that the virus has been proven to cause. On the other hand, positive results are usually very inaccurate for people who have no symptoms. In other words, the virus can be isolated from some very large fraction of positive testing people who express the associated symptoms, but only from a small fraction of positive testing people who express no symptoms. Thus positive tests in healthy people usually don't indicate active infections.

velope, along with gp160; Papadopoulos cites references showing that p32 is the "Class II histocompatibility DR" marker found on all human T immune cells.

(4) The existentialists hypothesize that reverse transcriptase is a constituent of HIV, and is used to make HIV DNA from HIV RNA; Papadopoulos cites references showing that this enzyme is a normal constituent of all human cells, and even some ordinary viruses, like hepatitis viruses, which are common in AIDS patients.

(5) Papadopoulos shows that no complete "HIV" RNA mol-

## Antigens and antibodies

One measure of the immune system's response to a substantial viral infection is the production by *B-cells* of proteins called *antibodies*. Antibodies latch onto and neutralize other proteins.

Proteins that elicit an immune response are called *antigens*. Viral antigens tend to be those proteins that compose the inner core, and those that decorate or line the outer membrane envelope. These are the proteins that the immune system "sees," whereas proteins inside the core—the viral enzymes—are shielded from immune surveillance. The immune system does not respond to non-protein molecules, like RNA and DNA.

ecule or DNA genome has ever been identified, that what is claimed to be the "HIV" genome represents bits and pieces of genetic sequences cobbled together, that the "HIV" RNA and DNA haven't been shown to code for what are claimed to be the HIV proteins, and that all the "HIV" genes are very similar to genetic sequences common to all humans.

(6) The existentialists hypothesize that the retrovirus-looking objects in electron micrographs of heterogeneous samples from AIDS patients are identical retroviruses, HIV, that consist of the "HIV" proteins and RNA abstracted from those samples; Papadopulos explains that since those samples are heterogeneous, there's no way to match the retrovirus-looking objects to any material abstracted from the samples, that retrovirus-looking objects are common products of stimulated T-cells, and that such objects are not necessarily viruses of any sort and can be proven to be so only when examined as isolates.

### HIV antibodies as autoantibodies

Although the "HIV proteins" haven't been shown to be constituents of a virus, they *are* the constituents of the ELISA and Western blot antibody tests for HIV. If Papadopulos is correct that these are ordinary cellular proteins, why would humans express antibodies against their own cellular proteins, a condition called *autoimmunity*? And why would such antibodies correlate (however imperfectly) with AIDS conditions and AIDS risks?

The *Bio/Technology* paper argues that antibodies against actin, myosin, and p32 indicate exposure to those proteins donated by other people via injected blood products, unsterile needles, and rectally deposited semen. These factors nearly unify all American AIDS patients, *and* they are oxidative stressors. So Papadopulos proposes that oxidative stressors cause AIDS conditions *and* positive HIV tests, thus explaining the correlation between AIDS conditions

and positive HIV test results.

(Which is not to say that every positive "HIV antibody" test indicates autoimmunity or oxidative stress, or that autoimmune phenomena always cause disease, or that oxidative stress always causes "HIV" phenomena or AIDS conditions.)

In non-industrial regions such as those in Africa where lots of AIDS patients reside, Papadopulos shows that HIV antibody tests (the only sort of HIV tests used there) cross-react with antibodies against numerous ordinary microbes and parasites that are rampant there due to extremely impoverished living standards. AIDS conditions in these regions, she says, result from those cross-reacting infections, other infections common among impoverished people, and poverty itself.

### Proving causation: another need for isolation

Papadopulos' group published another 1993 paper, "Has Gallo Proven The Role of HIV in AIDS?", in the Australian journal *Emergency Medicine* (5:113-123). This paper presented much of the same data and arguments about the lack of HIV isolation of-

## Western blot antibody tests and ELISAs

Scientists construct Western blot antibody tests by transferring to paper some of the protein bands from a Western blot gel. These bands will react when exposed to fluid that contains antibodies against the proteins in the bands.

Another test called the *ELISA* consists of viral isolates for which the constituent molecules have been broken apart from each other, but have not been separated from each other by electrophoresis. ("ELISA" stands for Enzyme-Linked Immuno-Sorbent Assay, which describes how positive reactions are demonstrated chemically.) This makes ELISAs easier and cheaper to make than Western blot tests.

But ELISAs are not as accurate. People test ELISA-positive if their plasma contains antibodies against just one of the viral proteins, whereas Western blot tests consist of the proteins separated into different bands, so clinicians can see exactly which proteins react with a person's plasma.

ELISAs are usually used as screening tests. Since people who test ELISA-negative have antibodies against *none* of the viral proteins, negative ELISAs just as accurately identify uninfected people as do Western blot tests showing no reactive bands.

But positive ELISAs are not as good as positive Western blots at identifying people with active infections. This is because there is no such thing as a specific antibody. Antibodies against a certain viral protein may react also with proteins of another virus, or even non-viral proteins. So positivity for antibodies against viral proteins is not unambiguous evidence that a person has been exposed before to a particular virus.

However, people positive for antibodies against all the antigens of a particular virus are much more likely to have been exposed to that virus than someone positive for antibodies against only one or a few of the antigens. Yet each receives the same positive ELISA. Only a Western blot can distinguish these people. Proper validation studies show higher accuracies (fraction of positive subjects from which viral isolates can be obtained) for positive Western blot tests than for positive ELISAs. But since ELISAs are cheaper, Western blot tests are usually reserved for people who first test ELISA-positive.

### Papers by Eleni Papadopulos-Eleopulos, Valendar Turner & John Papadimitriou

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HIV activity (predicted by the new hyperactive HIV "viral load" model). But Gallo found neither. Cells declared "HIV-infected" lived happily ever after, and would produce HIV indicators only when prodded by artificial stimulants.

The Australians emphasized that no researcher since 1984 has improved on Gallo's very weak case for HIV as a cause of AIDS.

#### All antibodies non-specific

The *Bio/Technology* paper presented a long list of non-HIV agents that can cause positive reactions on HIV ELISA and Western blot antibody tests. This is very bad news for those tests.

HIV antibody and antigen tests are constructed from heterogeneous samples rather than isolates, and validated against each other rather than the isolation gold standard. Therefore their validity requires that HIV proteins and the antibodies against them be *specific*. That is, the proteins must be exclusive to HIV, and the antibodies that react with them must react with no other proteins.

Gallo and the other existentialists, Papadopulos explains, simply assume that their "HIV proteins"—and antibodies against them—always indicate a virus made from those proteins, and nothing else. They base this assumption on no data, and no wonder. Only isolation—which none of them has achieved—can demonstrate this sort of specificity. Furthermore, Papadopulos' list of cellular sources for each "HIV protein," and her list of non-HIV entities that cause reactions with "HIV" antibody tests, absolutely falsify the specific antibody ideal for HIV.

#### False positives

Papadopulos explains that there is no such thing as specific antibodies against any microbial agent. *All* viral tests (including properly constructed ELISAs and Western blot tests for properly characterized viruses) "cross react" with entities other than their intended targets.

This is why test accuracies must be established for different groups (those with and without symptoms and risks associated with the virus) using the gold standard (virus isolation from fresh plasma).

Properly validated virus tests are not undermined by a list of cross-reacting entities. If the virus can be isolated from the fresh plasma of 99% of the people with certain symptoms who test positive in validation studies, then physicians would have a 99% certainty that a patient with those symptoms who tests positive has an active infection.

The existence of cross-reacting entities becomes important only in circumstances of low accuracy. In the world of properly constructed and validated viral antibody tests, that means symptom-free people, and people who have been exposed to cross-reacting factors.

Virus isolations are rarely achieved in symptom-free people who test positive, which means the accuracy is low for apparently healthy people. The only sensible interpretation for positive results in healthy people is that these people have experienced,

ferred in the *Bio/Technology* paper. But where that paper examined the absolute requirement of viral isolation for constructing and validating viral tests, this paper examined the absolute requirement of viral isolation for demonstrating a causal relationship between a virus and a disease.

The Australians focused here on Gallo's 1984 papers, which they characterized as the most thorough to date. They argued that a virus can only be considered causal for a disease if:

(1) It can be isolated in every case of the disease from fresh (uncultured) plasma. Yet Gallo claimed to isolate HIV only from cultures, and only after stimulation with agents that cause inactive viral DNA (provirus) to produce viruses that might not be present *in vivo*. Furthermore, Gallo could only claim HIV isolation in 34% of the AIDS patients tested, and even then these claims were based not on real isolation, but on the observation of certain proteins, reverse transcriptase, and retrovirus-looking particles, though usually not all at the same time.

(2) Adding isolates of the virus to cultures of cells of the type affected in the disease in question results in behavior consistent with the disease. In the case of AIDS, that would mean adding HIV isolates to cultures of T4 cells and looking for either cell death (predicted by the original killer HIV model) or high rates of

### Northern and Southern blot tests

Southern and Northern blots from viral isolates represent pure samples of viral DNA or RNA. Scientists use the material in these samples to produce viral tests that react with viral RNA or DNA in patient fluids. To do this, they construct small DNA or RNA molecules, called *probes*, that complement segments of the viral DNA or RNA. To test patients, clinicians make Northern or Southern blots from patient fluid (usually plasma, depending on the virus in question) that has been treated so that any constituent viruses will be broken apart, exposing the genetic material inside.

If there is lots of virus in the plasma, a distinct band will appear in the gel at the location characteristic of the genetic material of the virus in question. Adding the probes will confirm that such a band consists of viral DNA or RNA. If only a small amount of virus exists in the plasma, the genetic material settling at the characteristic location in the gel will become detectable only after the probes are added.

sometime in the past, an infection that is no longer active (and is thus inconsequential), or they were exposed to cross-reacting proteins.

Before the introduction of HIV science, physicians did not test healthy people for viral infections, except for people with certain risks, such as recent exposure to someone with a confirmed infection. Validation studies can show a relatively high accuracy for positive tests in symptom-free people with such a risk. So it is rational to test such people. HIV tests are the only viral tests administered routinely to healthy people with no risks.

In the strange case of HIV and AIDS, though, even testing people in the AIDS risk groups is a dubious enterprise. This is because the official risks that define these groups (rectal intercourse, unsterile needle use, blood product injections, residency in impoverished nations), involve exposure to non-HIV factors that cause cross-reactions with these tests.

### Virologist Lanka supports Papadopoulos

The *Bio/Technology* paper influenced most reappraisers to question the validity of "HIV" tests, mostly on the grounds of cross-re-

### Viral load tests

Patients rarely ever have enough "HIV RNA" to yield a detectable signal on Northern blots of fresh patient serum. Thus the necessity to invent "viral load" testing, which employs the polymerase chain reaction, *PCR*. *PCR* generates millions of RNA or DNA copies out of an original undetectably few molecules.

AIDS reappraisers consider these tests invalid. The concentrations of HIV RNA these assays usually indicate—hundreds of thousands per ml of plasma—would easily show up on Northern blot tests. But it doesn't show up at all.

activity. Few seemed to appreciate that the isolation question was the real crux of the matter. The question of HIV's actual existence seemed just too big for most reappraisers to tackle. Then along came a young German virologist, Stefan Lanka, co-author of an academic paper that properly established the existence of a marine virus, *ectocarpus siliculosus*.

The British AIDS reappraisal magazine *Continuum* published in its April/May 1995 issue Lanka's exposition, "HIV: Reality or Artifact?" This was the first article for a popular audience explaining Papadopoulos' contention that HIV simply does not exist, and that the phenomena considered to indicate its presence have non-viral explanations, such as artifacts of the lab procedures applied to cultures made from the blood of AIDS patients. The next issue (June/July) included a fiery and detailed exchange between Lanka and Steven Harris, a physician who advocates the HIV-AIDS model. That article displayed two electron micrographs of properly isolated viruses: Lanka's *ectocarpus siliculosus*, and adenovirus type 2 (which cause common colds). Those two micrographs exclusively contained identical virus-looking objects. Harris presented a micrograph of what he called an "HIV isolate." Lanka pointed out that this micrograph contained, in addition to retrovirus-looking objects labeled "HIV," lots of microvesicles and "macromolecular debris." Therefore it was not an isolate.

### Antigen tests

During the early course of a substantial viral infection, the plasma contains lots of virus, and consequently lots of viral antigens, but very few antibodies against these antigens. This is because the immune response has not yet caught up with the viral activity.

Sometimes there is not even enough antibody to cause a reaction with ELISA or Western blot antibody tests, which contain the antigens. So scientists have developed tests that contain *antibodies* against viral antigens. These tests react with patient plasma that contains viral proteins. Antigen tests, then, are the inverse of antibody tests.

This exchange created such interest—and *Continuum's* editorial board was so persuaded by Lanka's argument—that the magazine in its January/February 1996 issue posted a £1,000 "Missing Virus Reward" for anyone who could produce a micrograph of a proper "HIV" isolate.

### Papadopoulos answers the first challenge

In April, 1996, the *National AIDS Manual (NAM) Treatment Update* published an editorial answering the *Continuum* challenge. *NAM* made no claim on the prize, conceding an absence of the micrograph specified by the reward. Instead, *NAM* argued against the need for such a requirement in establishing the existence of a virus.

Specifically, *NAM* rejected the Papadopoulos/Lanka objections to contaminating material in the available "HIV" micrographs. "...It's like saying that it is impossible to identify a German shepherd dog by its unique appearance," the article reasoned, "if

### Active vs. inactive viral infections

Cells with inactive, or dormant, infections have inside them viral DNA molecules, called *proviruses*, that are asleep. Sleeping proviruses produce no virus, and thus can cause no disease, since viral replication is what destroys or damages cells in the course of a viral disease.

Viral DNA goes to sleep when the host immune system gains the upper hand. Among the anti-viral molecules secreted by immune cells are substances that put viral DNA to sleep. When immunity is suppressed, the plasma levels of these substances diminish, and sleeping viral DNA awakens to start producing new viruses, which show up in the plasma.

Since cell cultures contain no immune systems, they contain none of these anti-viral substances. That makes them ideal nurseries for viruses. When cultures are made from cells containing dormant proviruses, the proviruses have their ideal circumstance to spring back to life and generate a maximum amount of new virus. Some proviruses awaken from dormancy only when stimulated by agents that promote viral activity. These sorts of viruses make very poor candidates for disease causation, for obvious reasons.

it happens to be surrounded by poodles."

In the May/June issue of *Continuum*, Papadopulos' team responded to the *NAM* critique with a remedial lesson in microbiology: "The analogy with HIV is more like someone who does not know what a German shepherd is but who looks at an aerial photograph of a zoo," and notes that some of the objects look like dogs, then "mince[s] up all the objects in the zoo," and presumes to know which teeth, claws, hair, hearts, and stomachs came from the objects that looked like dogs, and claims that those objects are some new breed deserving of a new name.

Instead, German shepherds have been carefully studied on their own, which is why they can be identified merely by their image, even in the midst of other dogs. Certainly a new breed of dog could not be declared—and identified by aerial photographs (the human scale equivalent of an electron micrograph)—without first studying one up-close (the human scale equivalent of viral isolation).

If isolates were obtained of the objects labeled "HIV" in micrographs of heterogeneous samples, and those isolates were shown to consist of a unique, exogenous retrovirus, then there would be a basis for pointing out these objects in heterogeneous samples and declaring them to be "HIV."

Until then, nobody knows what the objects purported to be "HIV" are in any of the "HIV micrographs."

### Duesberg demurs, Lanka describes

By the July/August issue, *Continuum*'s reward had increased to £25,000, and none other than Peter Duesberg wrote in to claim the prize. Conceding that there existed no such micrograph as that sought by Papadopulos and Lanka, Duesberg argued that existing data "exceeded the [Papadopulos/Lanka] criteria" for virus isola-

tion: the isolation of "infectious full length HIV DNA" from "HIV-infected cells," and the detection of this DNA in some T4-cells of nearly 100% of people who test positive for "HIV antibodies," but in nearly 0% of those who test negative.

In the same issue *Continuum* published rebuttals by both Lanka and the Australian team, which now included a fourth member, David Causer, Senior Physicist at the Department of Medical Physics at the Royal Perth Hospital.

Lanka surprised everyone with his "Collective Fallacy: Rethinking HIV." Leaving it to "the distinguished Australians" to provide "a detailed reply to the Duesberg claim," he leaped past that dialogue and into a novel assertion: *all* retroviruses are fictions, artifacts of the contrived laboratory conditions invariably used to find them. He described Duesberg as:

*limiting his objections to the relatively minor aspect of whether HIV could cause AIDS or not, whereas he really ought to have smelt a rat regarding the whole concept of retroviruses. ...Indeed, the extraordinarily artificial and circumscribed conditions under which reverse transcription could be induced in the laboratory should have alerted everyone to the extreme improbability of such exclusively laboratory conditions having any bearing whatsoever on naturally occurring phenomena.*

### The Papadopulos treatise

Papadopulos' rebuttal was an exhaustive exposition entitled "The Isolation of HIV: Has It Really Been Achieved? The Case Against," included as a 24-page supplement. She asserted that until a virus has been isolated according to the criteria required by the *Continuum* reward, its constituents—including genetic material and proteins—cannot be cataloged. So there is no basis for a viral explanation for this correlation.

Yet Duesberg has a point. How can Papadopulos and Lanka explain the high correlation between particular proteins (and antibody reactions to them) and the detection of particular

### The culture skinny

Viruses replicate in two sorts of cells, *in vivo* (those inside living organisms, such as people), and *in vitro* (those maintained in laboratory culture dishes). Virus isolation from human plasma demonstrates *in vivo* viral activity, and isolation from culture fluids demonstrates *in vitro* viral activity.

However, isolation from the fluids of a culture composed of donor cells can not demonstrate that the donor harbors an active infection. It only demonstrates that the donor cells contain proviruses that are active under culture conditions. Transferring cells from a living organism to a lab dish can permit sleeping proviruses to awaken. Only examination of uncultured tissue fluids can diagnose viral disease.

DNA/RNA sequences? This can not be a chance occurrence.

Papadopulos agrees. But she points out that isolating DNA does not equal isolating a virus, and certainly does not "exceed the criteria" specified by the reward, which represent, in fact, an official standard procedure for retroviral identification which was

discarded only to accommodate "HIV." Logically, there is no basis for concluding that an RNA molecule abstracted from a heterogeneous sample (even one containing retrovirus-looking objects), or a strip of corresponding chromosomal DNA, originates from a retrovirus. Such an assumption can only apply to RNA abstracted from a retroviral isolate (and only if that RNA is shown to code for the proteins abstracted from the same isolate).

To explain the "HIV" protein-RNA/DNA correlation, Papadopulos referenced studies showing that the correlation between the proteins and the genetic material was not quite as high as in the study Duesberg cited. Then she proposed that the "HIV DNA" in cellular chromosomes might result from the rearrangement (transposition) of a few normal cellular DNA sequences in response to oxidative stress caused by both the AIDS risks (street drugs, etc.) and the laboratory agents required to observe "HIV" phenomena.

Duesberg says this would require an improbable number of nucleic acid rearrangements ("recombinations"), one for each of the 9,150 bases said to constitute the HIV genome. Papadopulos says the number of required rearrangements is actually much lower, since each of the supposed HIV genes are already very similar to recognized normal human genetic sequences.

Is Papadopulos certain that oxidation-induced recombination explains the HIV protein-RNA/DNA correlation? No. She's simply convinced that this is more likely than the Duesberg-Gallo explanation, which is that the "HIV" genetic sequences originate in a retrovirus that carries with it the "HIV proteins."

To her, the viral explanation is fatally undermined by several facts: (1) heroic attempts to isolate such a virus always fail, despite huge financial incentives and numerous attempts to do so by an enormous army of scientists dedicated to "HIV," whereas far less interesting viruses are routinely isolated by much smaller, less-funded groups of virus hunters; (2) what is called HIV RNA and DNA comes in many sizes and varieties that *always* differ from each other (no two are alike, even when abstracted from the same patient), whereas viral RNA and DNA should be of uniform length and composition; (3) the lethargy that characterizes what is considered "HIV replication" excludes the possibility that replicative mutation can explain the wide HIV genetic variation; and (4) no one has produced a whole "HIV RNA" molecule or a complete "HIV DNA" strip, offering instead as the "HIV genome" cobbled together bits of genetic material.

Papadopulos notes that when "HIV DNA" shows up, it does so in only a tiny fraction of T4 cells. Duesberg's explanation is that this means HIV simply infects too few cells to explain any disease. But if HIV is so lethargic as to infect only a few cells, how can its amazing variability be explained? Papadopulos' hypothesis predicts wide variability: if "HIV DNA" originates from the rearrangement of normal cellular DNA sequences, then each one originates independently and separately in each cell where it is found. Various points of origin would result in a variety of recombination products: DNA strips of varying lengths and composition, and

## RNA and DNA viruses

Viruses carry in their core only one sort of genetic material, either RNA or DNA molecules. These molecules are called proviruses when they reside inside a host cell, outside the viral core. Proviruses direct the production of all viral components, even replication of themselves, in the manufacture of new viruses.

Except for retroviruses, viruses that carry RNA are always active, but viruses that carry DNA can be active *or* inactive.

This is because RNA constantly produces proteins when it is in contact with amino acids (protein building blocks) and ribosomes (enzymes that translate RNA molecules into corresponding protein molecules). In the viral core, viral RNA has no contact with amino acids or ribosomes. But inside a host cell, viral RNA has all the material it needs to produce new viral proteins.

DNA, on the other hand, can not be directly translated into proteins. First it must be transcribed into RNA by an enzyme called *transcriptase*. But DNA has the ability to regulate its own transcription. So DNA can be active or inactive, whereas RNA can only be active.

Enzymes called *reverse transcriptase* will reverse transcribe retroviral RNA into corresponding DNA molecules. Consequently, retroviruses share with DNA viruses the ability to be either active or inactive.

corresponding RNA molecules transcribed from that DNA.

Papadopulos stresses that her argument against the existential hypothesis of HIV does not require that her alternative hypothesis be correct. Since the existence of HIV is not a default hypothesis, we are not obligated to assume that HIV exists in the absence of a better explanation. To the contrary, until unambiguous evidence is provided for HIV—in the form of a proper viral isolate—explanations for the data are open to suggestions. As far as the Australians are concerned, the viral model has been thoroughly examined, and it comes up empty. It's time to propose and study some new ideas.

## The Duesberg-Papadopulos dichotomy

Papadopulos' advocacy of a non-viral explanation for microbiological phenomena labeled as "HIV" remarkably resembles Duesberg's advocacy of a non-HIV explanation for pathological phenomena labeled as "AIDS": (1) Duesberg explains that the HIV-AIDS correlation is not as high as it's made out to be; Papadopulos makes the same claim about the HIV protein-DNA/RNA correlation; (2) Duesberg shows that the microbiological data unqualifiedly exclude a role for HIV; Papadopulos shows that the microbiological data unqualifiedly exclude definitive evidence of a virus; (3) Both say we should therefore consider non-viral explanations; and (4) Duesberg says that even if the alternative hypotheses are ultimately falsified, the HIV-AIDS model is not consequently resurrected, because it fails all on its own; Papadopulos says the same thing about the HIV existential model.

The February/March 1997 *Continuum* carried a second appeal from Duesberg responding to the Papadopulos and Lanka rebuttals. The editors entitled the article, "Near Enough *Is* Good

Enough?" reflecting their sympathy for the non-existentialist position. Duesberg restated his conclusions that rearrangement of normal chromosomal DNA sequences was less likely than the viral explanation, and that the traditional virus isolation requirements advocated by Papadopulos and Lanka were outdated and, in any case, less rigorous than those which he said had been achieved by HIV.

This defense of HIV's existence recalls the arguments used against Duesberg's own proposal that HIV is harmless. Within that discussion, Duesberg shows that HIV fails to meet the traditional and logical standards of microbiology, including Koch's postulates. Advocates of the HIV-AIDS model respond by proclaiming those criteria are outdated, and offer new criteria which accommodate the HIV-AIDS model.

The Australian response is summarized in the title, "Why No Whole Virus?", and reemphasized points made in their previous exposition.

### Electron microscopy

More interesting was Lanka's second rebuttal to Duesberg, which included some new insights. Lanka expounded on the implications of a lack of "HIV isolates" despite dogged efforts. This should not be so for a virus that exists. Lanka writes:

*It has been long known that what "AIDS" researchers have presented as photos of "HIV" show normal cellular [microvesicles]... As those particles are designed, in contrast to viruses, for cellular use only, they are very unstable when removed from their context, and not able to be isolated and photographed in an isolated state. Viruses are stable because they have to leave cells or even the organism in order to infect other cells or organisms anew. Using centrifugation techniques it is no problem to separate viruses from all contaminating components and in doing so to isolate them—then photograph them, then represent their proteins and genetic substance in a direct way... Genuine viruses are so stable that it is easy... to photograph them directly as three dimensional particles in the [scanning] electron microscope without prior chemical fixation. In contrast [microvesicles] are so unstable they can only be photographed [with a transmission electron microscope, which requires they be] in a chemically fixed state... in very thin sections. All that have been shown to us as [micrographs of] "HIV" are ultrathin sections [that include what are agreed to be] cellular particles. ...*

Sure enough, the micrographs of proper viral isolates presented by Lanka in his rejoinder to Steven Harris were photographed with the scanning electron microscope, and thus showed—with high resolution and three-dimensional relief—the outer surfaces of the viruses. In contrast, the purported "HIV" micrograph presented by Harris was photographed by the transmission electron microscope in "ultrathin sections," producing flat, transparent, cross-sectional images with no surfaces and poor resolution. According to Lanka, viruses are hardy enough to be photographed either way, and ought to be, since one reveals the surface in great detail, and the other reveals important cross-sectional information.

But there exists no published scanned micrograph of anything claimed to be "HIV." Since there are billions of dollars and tens of

thousands of scientists annually devoted to the study of "HIV," it seems improbable that this could indicate an oversight. More likely the retrovirus-looking objects called "HIV" are, like microvesicles, simply too unstable for scanned electron microscopy and procedures that could otherwise separate them from all other objects into pure samples, which is to say—in Lanka's

### Virus counting

How many viruses do infected people have circulating in their blood? There is only one way to answer this question definitively, and it of course involves preparing an isolate from patient plasma, and counting the viruses in the isolate.

Scientists start by obtaining from the patient a fluid sample, which they *serial dilute*. Serial dilution results in one undiluted sample, and several others of equal volume diluted by varying amounts. From each sample scientists prepare a viral isolate, which they view on a standard grid using an electron microscope. If the patient has a high viral concentration, the undiluted sample will contain too many to count, since the viruses will be stacked on top of each other, and overlap.

The viruses in one of the diluted isolates will be spread out enough so they can be counted accurately against the grid, which represents some fraction of the area occupied by the sample. By multiplying factors that account for the gridding and diluting of the sample, counting the number of viruses in the grid will yield the number of viruses present in the undiluted sample. Dividing this number by the pre-diluted volume yields the viral concentration (in particles per milliliter) in the patient's plasma.

This of course is too expensive and complicated for the clinical setting. So scientists can calibrate some of the viral tests to approximate viral concentrations. For example, the thickness and staining intensity of Northern and Southern blot bands are directly proportional to viral concentration. So is the staining intensity of antigen tests. So by examining these test results for patients who have had their viral concentrations established, scientists can derive numbers that convert band thickness or staining intensity into viral concentration.

opinion—they are too unstable to be viruses.

(Instability, by the way, gives the objects labeled "HIV" both the characteristics Papadopulos assigns endogenous retroviruses, the other being non-infectivity in their cell-free form.)

"'HIV' has never been identified as a secure biological entity," he concludes. "The logical explanation given that all the characteristics ascribed to 'HIV' are well-known cellular entities and characteristics, is that 'HIV' never was, and the claim of the existence of 'HIV' is not sustainable."

### On hemophilia-AIDS, T4 counts, and African AIDS

Papadopulos' contribution to the AIDS reappraisal movement transcends the discussion of HIV's existence. Remember that she

unifies all the proposed causes of AIDS, and even the agents required for "HIV" expression, by a common denominator: they all cause oxidative stress. She also shows that oxidation is a logical source of many diseases, including all that qualify as "AIDS."

In 1995 her team published a lengthy consideration of "AIDS" in hemophiliacs, "Factor VIII, HIV and AIDS: An Analysis of Their Relationship" (*Genetica* 95: 25-50). To their assertion that factor VIII contaminants cause AIDS conditions in both HIV-positive and -negative hemophiliacs, they also stress a point promoted by no other reappraising scientists: that there is not even a basis for HIV transmission via Factor VIII injections—or any other mechanism, for that matter—since what is called cell-free "HIV" is bare of the surface protein (gp160) supposedly required for infection.

The Australians have also advanced—with Bruce Hedland-Thomas and Barry A. P. Page joining Papadopoulos and Causer from the Medical Physics Department at Royal Perth Hospital—another novel hypothesis, this one refuting the role of lost T4 cells in AIDS. In "A Critical Analysis of the HIV-T4-cell-AIDS Hypothesis," they argue that the progressive drop in T4 counts observed in many AIDS patients does not reflect a loss of T4-cells. Rather, it indicates the conversion of many T-cells from producing T4 surface markers to producing T8 markers instead. Thus there is no need to propose a T4-specific factor, such as HIV, to explain AIDS.

Then there is the issue of AIDS in Africa, where the symptoms and proposed causes are often quite different than in the industrialized world. In 1995 the Papadopoulos team published "AIDS in Africa: Distinguishing Fact and Fiction" (*World Journal of Microbiology and Biotechnology* 11: 135-143), co-authored by PhD biologist Harvey Bialy, research editor of *Bio/Technology* who has spent a great deal of time in Africa. This paper attributes AIDS cases there to the same thing that causes identical symptoms (persistent fever, wasting, and diarrhea) in Africans who test negative: extreme poverty, featuring subsistent diets and rudimentary or nonexistent sanitation.

The paper also explores the implications of the very poor heterosexual transmission rate (one per thousand unprotected contacts with a positive person) assigned to HIV in the face of high fractions of African heterosexuals testing HIV-positive. Either African heterosexuals are much more promiscuous than their American counterparts, or HIV tests are especially problematic in Africa.

The Australians show that problematic testing is the more likely explanation. Malaria, tuberculosis, and other tropical microbes that are widespread in Africa feature proteins that elicit the same antibody response as some of the "HIV proteins." HIV proponents have not accounted for this in any of their experiments. They simply assume that Africans who test positive are indeed infected

by HIV, when these tests may instead be indicating very common and conventional infections.

### Gordon Stewart joins Papadopoulos

"It seems tragic," Duesberg said in one of his *Continuum* papers, "that over 99% of the AIDS researchers study a virus that does

### Tissue Culture Infectious Doses (TCID)

One of the ways that clinicians can characterize a viral infection is to approximate the plasma concentrations of *Tissue Culture Infectious Doses*, or TCIDs. One TCID is the minimum amount of virus required to produce viral activity in a standard culture of stock laboratory cells. Scientists determine viral activity by obtaining viral isolates, or by observing phenomena previously shown in isolation studies to be viral, such as the appearance of certain proteins.

To determine TCID concentrations, scientists take plasma from a patient and *serial dilute* it. Serial dilution involves producing from an original sample a sequence of samples, each of the same volume, but each one ten times more diluted than the previous. Thus going down the line from the original sample to the last, each will contain in relation to the previous one, a tenth of the plasma—and a tenth of the viruses—contained in the original sample.

Each sample is added to a separate standard culture. If the undiluted sample causes no replication, then neither will any of the diluted samples, and the plasma contains no TCIDs. If the undiluted sample does cause replication, then it contains at least one TCID. If the first diluted sample causes replication, then the undiluted sample contained at least ten TCIDs. If the second diluted sample produces no replication, then the original sample contained at least 10, but fewer than 100, TCIDs. This range can be narrowed down by now using a dilution factor smaller than ten.

Because the usual dilution factor is ten, this process is called *titration*, and the term TCID can be substituted with the term *titer* (or titre). In the above example, scientists would say that the original sample had 10 TCIDs, or a viral titer of ten. By dividing this figure by the volume of the original sample, they can calculate the plasma concentration for the patient, in TCIDs per milliliter.

Validation studies can correlate TCID concentrations with actual viral concentration. However, this usually is not done, because TCID values provide more important information than viral concentrations. Remember, only infectious viruses can cause disease, and only if they are present at concentrations great enough to cause a productive infection. So it is more important to know the concentration of TCIDs than the concentration of actual viruses.

not cause AIDS and that the few who don't are now engaged in a debate over the existence of a virus that doesn't cause AIDS."

Charlie Thomas, the retired biochemistry professor who used to teach at the medical schools at Harvard, John Hopkins, and the University of Michigan, takes a more popular view. "The debate over HIV's existence instigated by the Australians," he has said, "is the only issue of high scientific interest that has emerged from this HIV/AIDS mess."

The "HIV non-existentialists," as Duesberg calls them, acquired an important endorsement this year from the eminent British epidemiologist and physician Gordon Stewart, who is emeri-

tus public health professor at the University of Glasgow in Scotland. Stewart co-authored the Australians' latest paper, "HIV Antibo-dies: Further Questions and a Plea for Clarification" (*Current Medical Research and Opinion* 13:627-634), which argues that "the evidence for the existence of HIV and its putative role in AIDS must be reappraised." Voltaire, though, might side with Duesberg on this one. He said, "To not be occupied and to not exist amount to the same thing." And Duesberg and Papadopulos do agree on one thing. There is no HIV occupied with AIDS-causing activities.

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