Medicine

AIDS virus mutates to more lethal strains

by Warren J. Hamerman

An AIDS research report of uncommon scientific interest on the biological transformations over time in the HIV virus (the AIDS-causing Human Immunodeficiency Virus) was published in the journal *SCIENCE* of April 1, 1988 (Vol. 240, No. 4848, pages 80-82).

The report, whose senior co-author is the San Francisco researcher Jay A. Levy, appears under the title "Biologic Features of HIV-1 That Correlate with Virulence in the Host." Levy's co-authors were Cecilia Cheng-Mayer, Deborah Seto, and Masatoshi Tateno.

The report has extraordinary implications for both theoretical biology, public health policy, and research. With regard to public health, such findings make a compelling argument for universal testing (early and often) for medical pharmacological intervention into the asymptomatic individual to block the "self-improvements" in the virus, and a phased "Chicago-TB" model selective quarantine. They also strongly argue for emergency improvements in the nutritional level and basic living conditions of all mankind.

Blind alleys of molecular biology

It also explains why molecular biology research has run into one blind alley after another and has not succeeded in "catching up" with the disease. The virus, in short, has been the tortoise to molecular biology's hare in Zeno's paradox.

It is well known that people infected with HIV can be either asymptomatic or highly diseased. Numerous biologic, serologic, and molecular studies have shown that the HIV-1 virus is highly heterogeneous. Individual "isolates" of the virus can be distinguished by the different capacity to infect and replicate in a wide variety of human cell lines including T and B lymphocytes, macrophages, and brain-derived cells. But studies have shown a *varying* ability of the viral isolates to replicate to high titers or to induce cytopathic changes in these different infected cells.

Little has been known about the factors that influence progression from infection to AIDS.

The Levy experiments

The Levy study sampled viral isolates of four individuals over time. The viral isolates of the AIDS virus (HIV-1) obtained at intervals during the infection of the four, showed that the development of the disease correlated with the emergence of HIV-1 variants that were: a) more cytopathic; b) more able to replicate efficiently in a wide variety of different human cells.

In short, *the biologic properties of the virus appear to reflect its virulence in the host. Certain changes in the structure of the virus can influence its virulence in the host.*

How did the Levy group proceed?

First, isolates of the HIV-1 virus were obtained at intervals from peripheral blood mononuclear cells (PMC) of four subjects selected randomly from a group of seropositive individuals. They noted that the HIV-1 was recovered more readily from each individual as the disease progressed.

For example, in subject #2, the isolate emerged in the culture within 12 days, whereas the previous isolates in the same subject took one month to be detected when he was less sick. Then, they attempted to grow the isolates from the same individual in a wide variety of different human cell lines—T cells, B cells, macrophages/monocytes, and brain cells. The later isolates were *more cytopathic* in *more different cell types*.

Then, the biologic and serologic properties of the first isolate were compared with those of three other individuals. These isolates had been in culture for only three to four weeks. As the disease progressed in subjects #2 and #3, the HIV-1 isolate had a wider host range and greater cytopathic and replicative properties. For instance, in subject #2, the isolate obtained two (2) months before the patient died replicated quickly and to high titers *in all established human cell lines* and primary macrophages. In contrast, the isolate obtained six (6) months earlier from the same subject replicated with much slower kinetics and much lower titers in critical cell culture tests. The isolate from subject #3 did not replicate in any established human cell lines over a 30-day culture period.

When tested with three HIV-1 antibody-positive sera, each group of isolates from the same individual displayed similar patterns of sensitivity to serum neutralization. This tends to suggest that while the serologic properties of the virus remained stable, *the biologic properties changed.*

The isolates were purified and subjected to various specialized tests to study differences in viral proteins. *Only the viral envelope glycoprotein* (gp120) displayed variations, but these changes did not seem to correlate with differences in pathogenic properties.

They also analyzed infected cellular DNA. Although some

variations were detected in restriction enzyme patterns from each individual, the differences were limited when taken from the same individual over time, *but markedly different from different subjects*.

The Levy group summarizes their work as follows:

"These studies indicate that disease progression correlates with the appearance of variant viruses that are more cytopathic and have a wider host range than the original isolate. The variants we isolated could have coexisted in each host from the time of infection and had different levels of expression during the course of infection. Alternatively, the virus originally transmitted to the host could have undergone genomic changes during the course of infection. We do not believe these biologic changes reflect the conditions *in vitro* because, except for isolates from subject #1, all isolates were characterized within 3 to 4 weeks of isolation. If selection of one isolate occurred, then this preferential recovery gave the same results consistently: The three individuals who advanced in disease yielded the more cytopathic viruses than the individual who remained healthy. Finally, most of the isolates were retested after three months in culture and showed the same biologic properties. The lack of molecular change in HIV-1 after long-term passage has been reported.

"These results suggest that the development of disease symptoms in HIV-1-infected individuals is associated with the emergence of more pathogenic virus variants. Future studies with these isolates should provide information on the genes that determine the virulence of HIV-1, and identify potential targets for antiviral therapy."

The implications?

The virus is not a pure entity, but a self-evolving epiphenomenon of a disease process. Under certain conditions of gross devolution in the physical economy of the biosphere, a viral singularity is "thrown up" by the process itself. Without a continuous improvement of the most advanced species in the biosphere—namely, the human species as a whole—the process begins to cannibalize itself and transforms to a lower-level manifold of activity. The transformations thereby catalyzed take on a "life of their own," so to speak. In which, absent the intervention to reverse the biogeochemical crisis in the physical economy of the biosphere as a whole, the devolving process "favors" the replication of improved strains of the virus, its mutants, and its recombinants. These transformations are "mapped" or "projected" into the biology of individual infected human beings, who themselves serve as the "petri dishes" for growing ever more virulent strains of virus.

These transformations focus upon the very questions which initiated the famous 1974 Biological Holocaust study prepared by Lyndon LaRouche and his associates, which forecast the development and progression of an AIDS-like viral pandemic in the precise areas and time sequence that AIDS has followed.

The nonexistent food surplus of Europe

by William Engdahl

According to reliable sources from COPA, the Brussels central organization of European farmers' organizations, the European Community (EC) Agriculture Commission is privately admitting that the "surplus crisis" no longer exists, but publicly continues to act as though it does still exist at the "alarming" rates of the past three years.

One of the EC's main arguments to motivate the drastic farm austerity prices of the past several years has been the "soaring" cost of EC farm subsidies—fully 67% of total EC spending, $27 billion last year. Officials neglect to mention that that was how EC member governments initially wanted to have it.

The other factor willfully ignored by Brussels officials zealous to cut farm spending is that fully 33% of the increased cost for Brussels in the past two years comes from the dollar effect. Grain is priced in world trade, as are most agriculture exports, in dollars. As the dollar drops, the price the EC must compensate in intervention for exports increases. Nobody in Brussels will talk about this "non-agriculture" factor, despite the fact it is, according to one EC official, by far the largest cost increase factor in the last two years.

And now, with the Feb. 13 Brussels "Stabilizers" agreement of EC heads of state, the EC will automatically impose a complex set of punitive taxes and price cuts if even one ounce more than 160 million tons of grain is harvested in the EC. The "trigger" number was deliberately set at a level just above the extremely low 153 million ton harvest of the last year. EC farm sources expect this year's harvest to be above 160 million tons. Reliable EC grain trading estimates of the actual cost to the farmer of this new "stabilizer" are that prices paid to EC grain farmers of average size will be further reduced by at least 20% per annum for the period until the Single Europe Act goes into effect in 1992, establishing a single internal market.

Pointing to the above, EC Agriculture Commissioner Frans Andriessen told a Brussels audience at an April 7 food conference that the EC was moving toward a "market oriented" agriculture policy. That term, "market oriented," was coined by a multinational study made for the Trilateral Commission in 1985. It has become official policy of the U.S. Department of Agriculture and the EC. That is only further