Congo (9.7% for HTLV-1), and 0.9% in Senegal. While the percentages are not very high, they indicate the beginning of a dangerous overlap between the two diseases.

Johnson gave a second presentation on the subject of the impact of HIV on tropical diseases, during which he proceeded to establish a frightening forecast as to the future course of multiple epidemics in Africa and Ibero-America. He drew up a list of tropical diseases that would flare up because of HIV: leprosy, leishmaniasis, and also chagas.

Chagas, affecting millions in the Americas, has terrifying potential.

Sleeping sickness, could sweep Africa again.

Amebiasis and amebic meningocephalitis, can be expected to flare up.

Then schistosomiasis, lymphatic filariasis (elephantiasis), oncochercosis (one of the main causes of blindness), strongyloidiasis, all prevalent diseases in areas where HIV is breaking out—will make their appearance.

Johnson showed the common areas of prevalence of those diseases, as well as common areas of prevalence of HIV.

He then went back to the problem of trypanosomiasis. If we take the trypanosomiasis called chagas in Ibero-America, he explained, a disease transmitted by bedbugs, there is no really effective treatment. It affects several million people (8% of Brazilian children are infected in the northeast of the country). Chagas, which can lead to serious autoimmune diseases and is notably responsible for cardiac muscle diseases, can also remain latent in a majority of individuals.

When chagas affects an HIV-infected individual, it could evolve from the latent to the virulent form, said Johnson, "though I don't have a case to demonstrate this for you." During the question period, a French physician from St. Denis Hospital brought up a case confirming Johnson's worries. He stated that he had had a case of a patient from Honduras who died of AIDS with a virulent form of chagas disease.

Johnson also stressed the feared flare-up of the African form of trypanosomiasis, sleeping sickness, a disease that threatened to depopulate the continent earlier this century and which is again developing today, especially because of a collapse in insect (tse tse fly) control. He showed maps of tse tse fly presence, which show that one species of fly is prevalent in Western Africa where HIV-2 is spreading, while the other type is prevalent in Central Africa, where HIV-1 is most prevalent.

Johnson's presentation, unfortunately, corroborates the studies carried out by the Fusion Energy Foundation and the foundation's forecast as to the potential for a biological holocaust. I myself spoke on this subject during a conference in Munich, West Germany last year, and I used data similar to those of Dr. Johnson. I wish I had been wrong, I said to myself as the seminar ended. The emerging reality is too horrible to contemplate.

Experts disagree on AIDS fundamentals

One should not expect any more big breakthroughs in AIDS research, Dr. Robert Gallo of the U.S. National Cancer Institute announced at a press conference on June 6 in Montreal. He took an optimistic note: "There will be new findings, but the major things we need are done. . . . It's a problem of technology and time and testing this or that in a certain number of ways. . . . We probably have more information about how this virus works to cause the disease than we have about any single agent in the history of medicine."

Gallo proceeded to extol the promise of "soluble CD4"—what he has previously called the "magic bullet," the compound that it is hoped the HIV virus will bind to, instead of to the human cell. His enthusiasm overlooks the fact that many human cells that do not have CD4 receptors are still infected with HIV; that the infection of T4 is but one aspect of HIV pathogenicity; and that CD4 receptors do play a role which, once we inject a person with soluble CD4, could lead to important side effects, notably regarding immune functions.

Luc Montagnier of the Pasteur Institute in Paris did not agree with Gallo's boundless optimism. The pathogenesis of AIDS remains unknown, he said. "As long as we have no coherent hypothesis to explain AIDS, we shall have difficulties in developing rational therapeutics."

Dr. Michael Ascher presented his hypothesis on dysfunction of the immune system. His notion that the problem stems from overstimulation of the immune system, which exhausts itself, is conceptually much better than the standard "one virus kills one lymphocyte" version dished out by the WHO.

It were better to refer to the work of Elie Mechnikov, the Russian associate of Louis Pasteur and discoveror of phagocytosis. Mechnikov's basic tenet, that death is ultimately brought about by self-phagocytosis, or an autoimmune phenomenon seen in aging, is essential to the concept of immunity.

And what about the important and generally ignored fact of HIV-induced neurological dysfunction? (See *EIR*, July 1, 1988, "Should 'AIDS' be renamed 'CNSD,' 'Central Nervous System Disease'?" This topic will also be the feature of a future report in *EIR*.)