Over 70% of Ugandan children were vaccinated against tuberculosis, a disease which had been widespread throughout the 1940s and '50s.

With the advent of dictator Idi Amin in 1971, Uganda began its descent into biological holocaust. By 1973-74, health services were in a state of collapse. By 1974-77, the death rates in the counties of Rakai district began their takeoff, four years before deaths from "slim" were ever noticed. In 1979, the Tanzanian Army marched its way across the entire country. The invasion got rid of Amin, but also wreaked havoc on the Ugandan economy.

In 1980 and again in 1985, the areas around Lake Victoria were assaulted by epidemics of sleeping sickness (trypanosomiasis). "Uganda is the most serious situation in Africa regarding tryopanosomiasis," Dr. Peter de Raadt of WHO told *EIR* in 1985. "There is a complete breakdown of vector control in Uganda since Idi Amin's time. I was there in the 1960s and saw not one case of sleeping sickness." In 1985, the outbreak affected 20,000 people.

Today, in Uganda, there is one doctor for every 23,000 people; a mere 3% of the country's gross national product is used for health care. According to a health survey carried out in 1989 by the Ugandan Health Ministry, 46% of the children in rural areas are moderately to severely stunted, reflecting a general condition of chronic malnutrition. The mortality rate for children under five years of age is 180 out of 1,000—close to 20%. Of the children under five years surveyed, 41% were reported to have had fevers in the four weeks prior to the survey. "It should be noted," the report said, "that malaria is endemic in Uganda and therefore most fevers in children are attributed to malarial infection." Of the rural households surveyed, 1.7% had electricity and 0.0% had refrigerators.

The survey had been carried out with a grant from the U.S. Agency of International Development. The purpose was to discover how best to foster birth control in order to lower fertility rates.

But lowering fertility rates is hardly the problem in Africa. Condom distribution is hardly the answer to the AIDS epidemic. As Mr. As Sy, head of the Third World Project in Senegal, reported to the WHO conference in July, "Loads of condoms are being sent to villages where people are just lying there, already too sick or too old to have any use for them."

The case of AIDS in Africa exposes the truth of AIDS everywhere: The HIV virus is the result of the collapse of the physical economy under the dogmas of the IMF and British system free trade. Reversal of the AIDS epidemic requires 1) overturning of the malthusian-motivated lies of the WHO and CDC, and 2) full-scale mobilization to carry out the public health measures whose effectiveness history has repeatedly proven.

Any other approach constitutes criminal protection of the AIDS killer.

Interview: Dr. Mark Whiteside

'Safe sex' will not stop AIDS epidemic

Dr. Mark Whiteside of Key West, Florida, was one of the first to draw attention to the environmental factors associated with AIDS. His views were based on work he carried out with Dr. Carolyn MacLeod and the Institute of Tropical Medicine in Florida, on AIDS cases in poor neighborhoods in Belle Glade and Miami, Florida. The implications of this work have been systematically stifled by the Centers for Disease Control and the Centers' insistence that AIDS is almost exclusively a sexually transmitted disease. Linda de Hoyos interviewed Dr. Whiteside on July 27, 1992.

EIR: You have done a lot of work with patients with AIDS since 1988. Do you still stand by your view that AIDS is basically a "tropical, environmental-based, probably insect-transmitted disease, with secondary blood transmissions"?
Whiteside: Yes.

EIR: When we talk about AIDS at this point, what do we mean by AIDS? What would be a diagnosis?
Whiteside: AIDS still remains the same. It is a defective cellular immunity where it reaches a point that you develop opportunistic infections, or Kaposi's sarcoma. That's what we call AIDS. The definition might change to the point that anyone with antibodies to HIV and less than 200 total T-cells would as qualify as having AIDS. But that has not formally happened.

EIR: Do you consider that there is a major difference between infection with HIV and AIDS? Is HIV necessarily causal to AIDS?
Whiteside: I never thought it was the only cause of AIDS. I've accepted it as the most important marker for the disease. You can get exposed to HIV and make antibodies in a few weeks to a few months, and it may be 5, 10, 15, 20 years before you come down with full-blown AIDS.

EIR: The African "slim disease" seems to have a very rapid onset. There are immediate symptoms which are recognized as AIDS—dry cough, diarrhea, herpes zoster. Is that similar to AIDS in the United States?
Whiteside: AIDS varies a little bit depending upon geography. In the tropics, there is more wasting and diarrheal dis-

ease. There is a certain increase in what are called tropical infections. Where you live makes a difference, obviously. They are similar diseases, in that it is a breakdown in the body's acquired immunity over time. It is going to have different manifestions. Most of the opportunistic infections that one falls prey to if one has AIDS have a reservoir in the environment. It depends upon what environment you live in.

EIR: The AIDS epidemic began to emerge in Africa in 1981. This is also the case for the United States. Do you have any idea as to how long AIDS or HIV has actually been around?

Whiteside: Obviously something happened in the late 1970s, to create an epidemic situation. The cat was let out of the bag, so to speak. A milder form of the disease has been in these endemic areas for a long, long time. We always thought there was a relationship to endemic Kaposi's sarcoma, for example. That's why we looked to Africa as a possible starting point. We were one of the first to do that. However, some of the arboviruses that I am looking at and have found in some of my patients are Latin American agents. So I am not so sure that there has not also been an AIDS locus in Latin America. There's the case of the kid in St. Louis who had HIV and Kaposi's sarcoma in 1967. Worldwide there are foci of Burkitt's lymphoma and Kaposi's sarcoma going back over the centuries. So, this type of thing may have existed in a milder endemic form in endemic areas.

But we know that epidemics of the disease occurred in North America and Africa almost concurrently, which is interesting. The epidemic in Asia was the last to take off.

EIR: You postulated a relationship between AIDS and arboviruses—mosquito-borne viruses.

Whiteside: That is by no means proven. It was just our area of interest. I believe in co-factors. I think that things are necessary to activate HIV, because it doesn't do anything unless it is activated *in vitro* and probably *in vivo*. We began looking at the role of multiple infections leading to immunosuppression in the tropics. We became more focused on insect or viral infections which are known to be potentially immuno-suppressive—can wipe out the lymphoid tissue altogether.

Secondly, we found some interesting articles which reported that arboviruses—arthropod-borne viruses—can activate or stimulate the growth of *animal* retroviruses. So it is a dynamic type of system.

You can put Venezuelan equine encephalitus (VEE) in a three-week-old mouse, a presumably virus-free mouse, and later look inside that mouse and find that it is full of Type-C retroviruses. There you have stimulation of growth of an endogenous retrovirus that is simply in the genetic code of that suckling mouse.

On the other hand, you can take a different type of arbovirus, like guaroa virus, and put that in a mouse with another type of retrovirus, a type of leukemia virus, and you will greatly enhance the growth of that retrovirus, that leukemia virus. That would be the stimulation of the growth of an exogenous virus.

So, we were looking at that type of dynamic interaction between an insect-borne virus and a retrovirus.

We also looked at equine infectious anemia. We know that to get insect-transmitted infectious anemia in horses, so-called swamp fever, you need special conditions. You have to crowd the horses together. You have to have a lot of large, blood-sucking flies. The final thing that you need, which veterinarians could never figure out how you got, was very high levels of viremia. We postulated that underlying arboviral infections—epidemics of arboviral infections—trigger these retroviruses' infections, and that is the means by which you get high levels of retroviruses.

It is also interesting that arboviruses tend to go across animals, whereas retroviruses are very species-specific. Humans don't tend to get feline leukemia, or bovine leukemia. We have our own retroviruses.

EIR: In 1988, you mentioned a 1962 study which linked the arbovirus bunyamwera fever to Kaposi's sarcoma.

Whiteside: These people who were working back in Africa in those days were convinced that these tropical tumors—Burkitt's lymphoma and Kaposi's sarcoma—were environmental and possibly insect-transmitted. They went so far as to feed tumor suspensions to *Aëdes aegypti* mosquitoes and try to get transmission to experimental animals. Traditionally, those tumors have been linked to environmental factors—climate, rainfall, altitude—to the tropical belt of Africa, not to some sexual disease. As one of those environmental links, these doctors looked at insect-borne viruses. Using the method they had at that time, hemaglutination inhibition, they tested a panel of insect-borne viruses that were prevalent in central Africa, the area they were testing. They found some interesting results with bunyamwera fevers—probably a stem virus from Uganda. They found that compared to controls, a third of their Burkitt's lymphoma and Kaposi's patients had antibody to bunyamwera. But what was very interesting is that something like two-thirds of the parents of those patients with those tumors had antibodies to bunyamwera.

The other point would be, that used only one testing procedure, and there are several different bunyamwera viruses in Africa.

EIR: Do these types of arboviruses like bunyamwera also exist in North America?

Whiteside: Yes. Arboviruses are worldwide; there are hundreds of them. Maybe only a hundred of them cause disease in humans. You divide them into groups based on the serologic reactions and their morphological characteristics. There is Group A, and Group B. But the largest single group of insect-borne viruses is the bunyaviridae family. But in that family

is bunyamwera surrogate, which we are looking and it has 20 members worldwide—7 in North America, 7 in South America, 5 in Africa, and only 1 in Asia.

They are all over the United States. In our studies in south Florida, we were looking at Tensaw, which is the southeastern United States bunyamwera virus, and found antibodies in a number of our patients. And we were also looking at Maguari, which is the Latin American bunyamwera representative. It was isolated from Jamaica in the past, and also in Brazil. It is the one that the Centers for Disease Control, in their study which was guided by us, found in a high percentage of persons in Belle Glade, Florida, neutralizing antibodies to Maguari. We could speculate that it was brought in by the tens of thousands of Jamaican sugarcane workers that they bring in every year. But the *American Journal of Tropical Medicine and Hygiene* tried to claim that since Tensaw and Magauri are so close, that what they really found was a strain of Tensaw, that it was not a type of virus that had been introduced from outside.

That brings you to an area of controversy. Many scientists feel that these viruses tend to be somewhat ecologically bound. This means that a virus coming from Africa would not establish itself, in the ecology here. There could be epidemics of it in Africa, and people coming from Africa with the disease coming here and spreading it to a few other persons, but the virus would not establish itself.

My view is that these viruses could be introduced or reintroduced if the conditions were right.

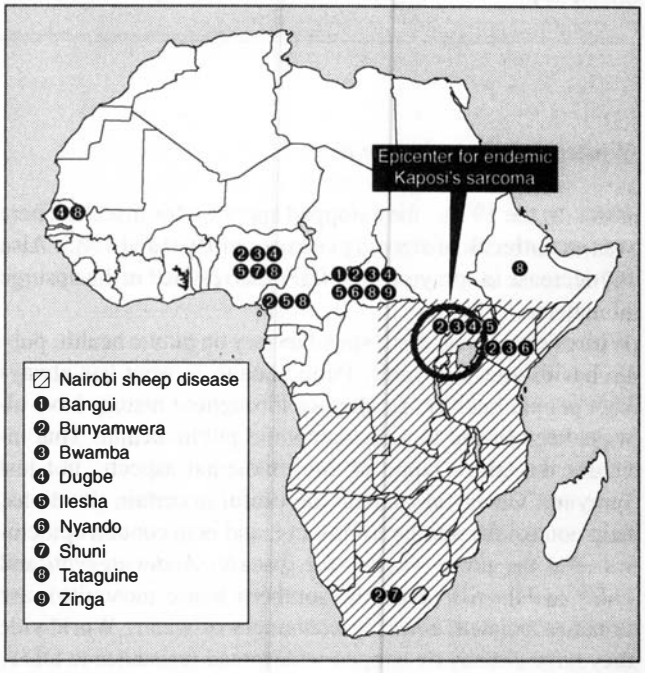
Our view was that if you brought patients from bad conditions in Haiti to bad conditions in the United States, then you would get trouble. But if you brought them from bad conditions in Haiti or Jamaica to good conditions in the United States, these diseases should be expected to gradually go away. This is the interesting thing with Haitians. AIDS in Haitians was beginning to go away in New York and Montreal years ago, and in more recent years, it has begun to go away in Haitians living in decent conditions in Miami. But it hasn't gone away among poor Haitians living in Belle Glade, Florida, or poor Haitians living in Haiti. Now explain that to me on the basis of a sexually transmitted disease.

EIR: In Africa, there are already so many factors suppressing the immune system, including malaria.

Whiteside: Of course, they tried to discount any relationship to malaria and HIV in the early days. Because some studies suggested a correlation in antibody studies between malaria and HIV, and that was discounted. The only point that they offered was that children with recurrent malaria infections get anemia and they have to get blood transfusions, and that brings in the tainted blood factor.

A major article in the *Journal of the American Medical Association* a few years back did accept that multiple infections in the tropics—malaria, filariasis, schistosomiasis, trypanosomiasis, the list just goes on and on—play some type of role. It is interesting that the one major group prevalent in the tropics is the insect-borne viruses, which they pretty consistently have not looked at. This goes to the crux of our so-called theory.

FIGURE 1
Distribution of bunyaviridae family arboviruses in Africa



Source: *Hunter's Tropical Medicine*

In the tropics you get multiple infections. These insect-borne viral infections may cause some symptoms, but not much; they are silent killers. Arboviruses cause no symptoms, or if they do, it is an uncharacterized febrile illness. On occasion, they can cause encephalitis, or hemorrhagic disease. But characteristically, they don't. But then with repeated exposure, you get worse disease through immunity enhancement of infection, where low sub-neutralizing level of antibody actually makes exposure to a second, related agent far worse. Something goes awry and at some point, retroviruses may also be activated.

I do take retroviruses very seriously. I work with antibodies with HIV every day. I take it as the most important marker we have. It may well contribute to immuno-suppression once it is activated. But I am still interested in the background immuno-suppression.

I think there are causes of AIDS that may come before HIV.

EIR: Come before it?

To think you can control an epidemic by putting up some posters and handing out condoms is, I think, the height of absurdity. This is the "blame the victim" concept, and you can then ignore the breakdown of public health.

Whiteside: Come before it.

EIR: In the 1970s, they stopped spraying for insects. There was an outbreak of sleeping sickness in 1980 and 1985. Also the decrease in spraying with DDT has resulted in the upsurge of malaria.

Whiteside: If you don't spend money on public health, public health goes downhill. Public health is what has always kept people healthy. Epidemics throughout history have always been linked to a breakdown in public health. That includes the entire range of environmental aspects, not just spraying. Chemical controls are useful in certain situations, help control the number of insects, and help contain epidemics. But the urban vectors for disease, *Aëdes aegypti* and *culex quinquefasciatus* (the southern house mosquito), are urban-associated, breed in containers of water. Worldwide they have already developed widespread resistance to DDT, malathion, whatever. Spraying really does nothing to control insects like this, which are the most efficient vectors of viral agents.

The whole key to control there is decent housing, sanitation, control of breeding, public education.

EIR: What are your thoughts on this report from the recent Amsterdam conference on AIDS, that people with AIDS are testing negative to HIV?

Whiteside: They are hypothesizing that this is some different strain they can't pick up—another retrovirus, HIV has mutated or something. But that is because they strongly believe that HIV is the cause. I don't necessarily adhere to that view. My natural assumption is that there could be a completely different agent in those patients. If you can't find antibodies by any technique, and you can't find virus by any culture technique, or PCR [polymerase chain reaction, in which a latent virus is stimulated to produce gene product to force detectable levels of the antigen], or any of the sophisticated methods that we have to detect viral antigen, then it's not there. There could be some other agent causing immunosuppression—something we haven't been smart enough to find yet.

Part of the reason I think that arboviruses could have something to do with the development of AIDS is that I believe what I can see with my own eyes. In the early days, a fellow at the CDC published an article on an unidentified viral particle in an intestine of a patient with AIDS. This was before HIV was identified as the cause of AIDS. He was going through the entire body tissue looking for a virus, with the use of an electron microscope. It might be like looking for a needle in a haystack, but it is a logical thing you would do, also. And he found this viral particle.

I thought this viral particle had all the appearance of an arbovirus. Even in a bunyaviridae-type virus, they reproduce by going into the endoplasmic reticulum [area of membranes within the cell], and when they form their viral envelope, they actually use the endoplasmic reticulum to do that. They form out of there these round swirls and form interesting pathologic structures.

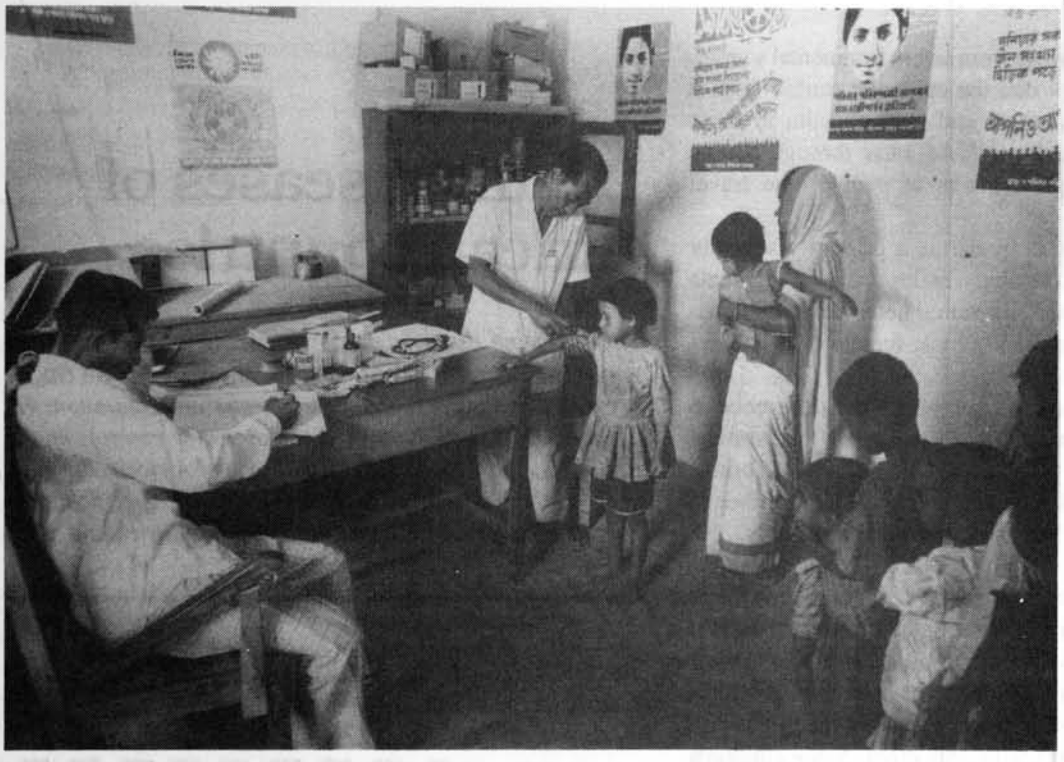
Because I was impressed with the similarity and also because I was looking at arboviruses at the time, I went to great pains over a couple of years, to get the reagents to specifically stain that tissue where he found that unidentified virus with antibody to a representative bunyamwera virus (we were looking at Tensaw and Maguari), and the AIDS tissue that we stained was strongly positive by fluorescent staining method for bunyamwera virus, whereas control tissue on all controls we could do, was negative.

You could argue that it is just there, and these viruses are prevalent worldwide, and we all have them in our gut. That could be true. Just because it is there does not mean that it is involved in the etiology. But there is supporting evidence from what we know about these viruses and their potential to cause disease and these tumors—Kaposi's sarcoma and Burkitt's lymphoma in Africa. There is also the fact that we are looking very closely at hepatitis C, because morphologically it greatly resembles a Toga virus, or potentially some member of the arbovirus family. It is a small RNA virus and morphologically looks like Toga virus. I think there may be an association between Type C hepatitis and AIDS in some of these areas. They may be transmitted in a similar fashion, not just by blood means.

I remain interested in an arbovirus as a potential co-factor in a disease, as interacting with a retrovirus.

EIR: What would you propose as a line of investigation?

Whiteside: If I had the time and resources to do it, I would go back to looking for a virus in that tissue where they found this virus. I would point out, though, that they found other viruses in that tissue. Our gut has a lot of viral agents. I would test AIDS patients for antibodies to an entire panel of arboviruses, sometimes based on serological group or geo-



A village health clinic in Bangladesh during the 1970s. "If you don't spend money on public health, public health goes downhill. Public health is what has always kept people healthy. Epidemics throughout history have always been linked to a breakdown in public health."

graphical area, using different serologic techniques, because I don't think that you are just talking about neutralizing antibody activity, especially with the whole issue of enhancement. Studies on viral ultra structures [structures created by the virus itself within tissue] should be carried out.

Field studies should also be done, where you go into an area of high AIDS concentration, like we did in Belle Glade, and study the insects not only for the presence of arbovirus and also for the presence of retroviruses, for HIV. That brings up the whole issue of the potential for mechanical transmission of retroviruses, given that special setting—overwhelming crowding and so forth. That was never done. We made a weak attempt a number of years ago to look for an arbovirus. No one, to my knowledge, has ever looked for HIV in insects in south Florida.

It would also be very interesting to give animals combinations of arborviruses and retroviruses and see what happens.

EIR: It seems to me that the reports that AIDS has already swept through large parts of the rural areas of central Africa, seem to corroborate that it is an environmental disease.

Whiteside: That's the whole key. We were accused early on of fostering panic by talking about mosquitoes. It happens that insect-borne viruses that I am interested in are mosquito-transmitted. But a number of other arthropods can potentially transmit arboviruses. But all we were saying from those very early days, and the whole formulation from Belle Glade, was that environmental factor has something to do with this disease. It can't help but have something to do with this disease, not only in the development of the disease, but in the progression of the disease. Anyone with low immunity sent out to live in terrible conditions is going to get sick faster.

That has been borne out by very recent data which I just read in the paper this week, where they were saying that patients with AIDS that had shelter and medical care and so forth might live an average of three years, with full-blown AIDS. Homeless patients with AIDS die within nine months.

EIR: It's the same thing in Africa, six to nine months.

Whiteside: Poor patients in Belle Glade lived only a few months. Down here, middle-class gay men with AIDS often live up to five years. This is with full-blown AIDS. In terms of progression, the environment almost certainly plays a role. The vast majority of these opportunistic infections are either reactivated diseases, things that we have all been exposed to like pneumocystis, or that you can get from a bad environment. TB is right up there, and cryptococcal meningitis.

My formulation remains the same. AIDS is a tropical-based, blood-borne disease. I don't know that I would even try to claim that it is first and foremost an insect-transmitted disease worldwide. I claim that it is a blood-transmitted disease. I don't know if anyone could argue on that point. HIV per se fulfills none of the classic criteria for a strictly sexually transmitted disease—not one. It is a blood-transmitted disease which can be secondarily transmitted by sexual practices that break the skin, the so-called indirect parenteral route. But that is a blood means of transmission.

I do think that AIDS arose from an environmental source, and I hold to the concept that the epidemic started from a breakdown in the environment and public health in these endemic areas. It spread from those areas through people traveling, given the large numbers of people who travel worldwide.

I do not believe that it spread as a primarily sexually transmitted disease. I do strongly believe that these agents can be sexually transmitted, including heterosexually transmitted. I think it is more readily male-female than female-male disease.

I don't think that, however, explains the difference in saturation in endemic areas. They try to explain that with the co-factor of other sexually transmitted diseases, which brings you to the point that all Africans with AIDS are either a) sexually promiscuous or b) have sores on their genitals.

EIR: Since the environmental factor is ignored, measures are not being taken that could control the disease. I recently read a book on AIDS in Africa which went so far as to suggest that it was unsafe for any Ugandan to have any sex at all!

Whiteside: My view is that there is only so much you can do about people having sex with each other. Since condoms are not 100% protection, if you have sex with an infected person, you could still have potential disease transmission. Most African men don't use condoms anyway. To think you can control an epidemic by putting up some posters and handing out condoms is, I think, the height of absurdity. This is the "blame the victim" concept, and you can then ignore the breakdown of public health.

We have been accused of trying to draw attention away from safe sexual practice and those issues, and that is just a total fallacy. I have always accepted potential sexual transmission and we had one of the first AIDS screening clinics in the United States, long before there was a test for HIV, in 1982, and we always counseled on sexual practics—avoid anal intercourse and all the rest. This was long before it was fashionable. To this day, there's not a day that goes by, that I don't counsel about so-called safe sexual practice.

If the environmental factor were recognized, there would be a lot more you could do about AIDS. It would be everyone's concern. But it would mean more money, and I think that's the so-called hidden agenda.

EIR: But public health is cheaper in the long run.

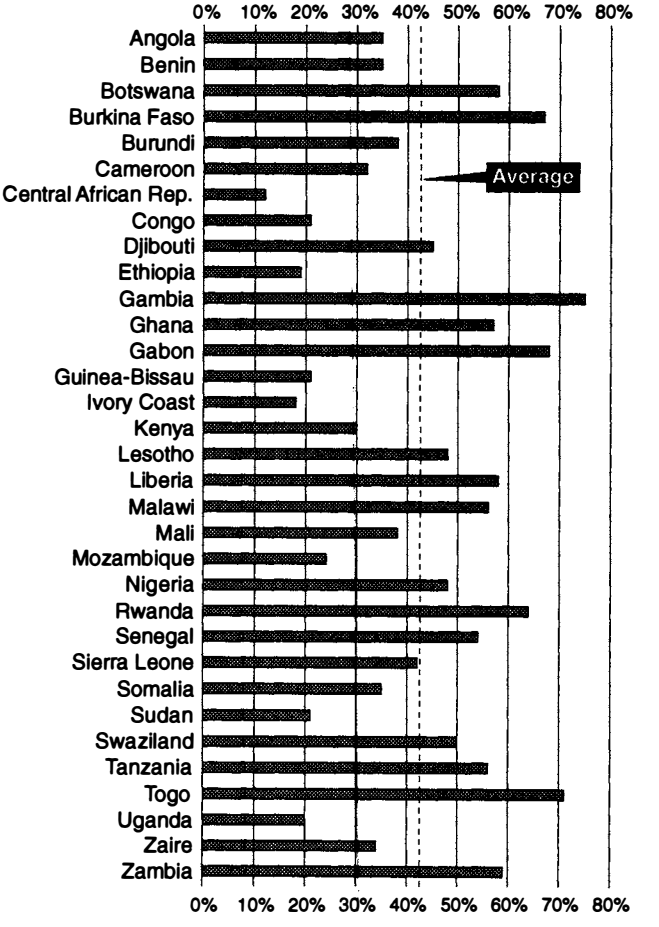
Whiteside: There has to be a decision to make public health a first priority again. I don't know what it would take to make people wake up to the fact that we have to protect our public health. You know the old saying, "You need a good, old-fashioned epidemic to make people wake up to maintain public health." In my view, we have that good old-fashioned epidemic now. Tuberculosis would be a good example, and people are still not fundamentally changing the model.

Deadly diseases of African countries

The following diseases are listed by African countries as major health problems. As can be seen, most of them can be prevented by the presence of clean water and sanitation, the usage of DDT and other methods of vector control, or the use of vaccines. Nevertheless, millions of Africans die each year of these diseases. Source for disease specifications: Hunter's Tropical Medicine, *by G. Thomas Strickland.*

FIGURE 1

Percentage of population with access to safe water in sub-Sahran Africa



Source: UNDP, 1992.