***This is an oral history interview on the NIH response to AIDS with Dr. Thomas C. Quinn on 5 December 1996. The interview was conducted in Dr. Quinn's office at the Johns Hopkins University School of Medicine, Baltimore, Maryland. The interviewers are Dr. Victoria Harden, Director, NIH Historical Office and the DeWitt Stetten, Jr., Museum of Medical Research, and Dr. Caroline Hannaway, NIH Historical Consultant.***

Harden: Dr. Quinn, we would like to start these interviews by asking you to describe briefly your personal background, where you grew up, where you went to school, and the positions you held before you came to the NIH.

Quinn: I was born 11 May 1947 in New Rochelle, New York. I attended the University of Notre Dame in South Bend, Indiana, and received my bachelor's degree and then went on to earn my master's degree in parasitology. Since I wanted to spend more time taking care of patients in addition to working in a research laboratory, I earned a medical degree at Northwestern University in Chicago, Illinois. My research interests solidified during graduate school and medical school to include parasitology, particularly malaria-type research and the vector spread of filariasis. This was the beginning of my interest in epidemiology, but from the perspective of how the disease spread via an insect.

I completed my internship and residency in internal medicine at Albany Medical Center in New York in 1977. By then I had my bachelor's, master's, and a medical degree, and was board-certified in internal medicine.

Clinical practice was an option at that point, but I was still drawn to laboratory research, particularly clinical research, and I still had that burning desire to work on malaria. So I went to the National Institutes of Health in 1977 and joined the Laboratory of Parasitic Diseases. For the next two years Dr. Lou [Louis] Miller and Dr. David Wyler mentored me solely on immunology and malaria research. It complemented what I had done during medical school.

Harden: Before you go on, let me ask you to elaborate a little. We like to ask physicians in general what forces or influences convinced them to go to medical school. You indicated that you had been in parasitology and then decided to go to medical school rather than aiming at medicine from boyhood. Could you elaborate? What was so attractive about malaria and parasitic diseases? This is a very unusual field.

Quinn: It is unusual. I actually was interested in medicine during my college days, but I was torn between the two routes. Do I go and do graduate research solely, or do I go directly into medical school?

I signed up for a course in parasitiology with Dr. George Craig, who has since

passed away two years ago. He stayed at Notre Dame his whole career, and he was the most enthusiastic person. He excited you about the possibilities of his type of research.

When you think about parasites and how they get into human beings and how they can take over—a red cell in the case of malaria; in the case of worms, in the intestine or migrating through lungs—it is absolutely fascinating that these organisms coexist with man, so that they can thrive and reproduce themselves and spread to another person. The parasites have worked out these mechanisms over centuries.

I would say it was Dr. Craig who got me interested in malaria and in parasitology. During my graduate year, he taught a course called tropical medicine, and that is what got me interested in the whole gamut of tropical diseases. There is always someone who touches you in your years of development, who angles you in a direction, and I would have to say he [Prof. Craig] had a lot to do with that.

On the medicine side, if there was someone who influenced me—and I think there was—it would be my uncle, who was a physician. He was an internist in private practice and never did research per se. He was a cardiologist. But I was close to him, and he used to take me into New York Hospital. He practiced at Cornell [University Medical Center] and was a very respected clinician. I liked biology, and this was medicine and biology fitting together, and I said, "I will give that a try as well."

It was during the year that I was in graduate school that I decided to get a medical degree. I could just have stayed in the laboratory and gotten a Ph.D., and I was offered the chance to do that through a Public Health Service training award. But I felt I could do more in the area that I wanted to work in, which was international health, tropical medicine, if I had a medical degree.

But then I missed the research part. So for two years, I worked at a bench once again, just like I did in graduate school.

Lou Miller, whom you know is head of malaria research, just received the Bristol Award, a very famous award, last year in New York. What a great mentor he was!

Harden: Do you want to elaborate on the research you did during those two years at the NIH?

Quinn: When I got to the NIH, my two mentors encouraged me to learn a little about the

immune response and how it deals with malaria. My immediate supervisor was David Wyler, who is now a professor at Tufts [University]--and Lou Miller. I dealt with an animal model, rats specifically, which got a malaria parasite called *Plasmodium burgeii*, that gets inside a red cell.

Right at this time, someone else in another laboratory working on another disease showed that you could label these red cells with a radioactive label trace, put them back into the animal, and then you could monitor the clearance of that infected red cell. No one had done it with malaria before. They had been doing it with other types of autoimmune diseases. I did it with malaria. It was the first time that had ever been done. Once the malaria parasite gets inside the red cell, the questions are how long does it last there, and where does it go. What I tried to discern was the natural history of an infected red cell with malaria in it. You could then manipulate the system in the laboratory. You could infuse immune sera and see that it clears that infected cell very rapidly. It showed that humoral antibodies were very important. If I took the spleen away from the rat, it could not clear the infected cell and the rat died. So it showed that the spleen was absolutely essential to the contribution of the immune response to the malaria parasite.

My first 10 or so publications reflect that early work. But it was basically the biology of malaria inside an erythrocyte, a red cell, and how the host—in this case, the rat—dealt with that particular infected cell.

Harden: This also reflects, does it not, that this was a fairly early period in the understanding of immunology at the molecular level?

Quinn: Very much so. At that point, we had not gotten very molecular. That was to come along in the subsequent years. But this research helped lay the groundwork for a better molecular understanding of the biology of malaria and for how to develop a better vaccine. I did not get into vaccines, but I hope that some of my work was instrumental in laying some basic building blocks towards what is now being tested in the field, a series of malaria vaccines.

The importance of malaria is that it is one of the leading causes of death in developing countries in young children. We will get to AIDS a little later, but when you ask is AIDS a leading cause of death, it is, but from ages 15 to 49. Usually you cut the age groups. If you ask, what is the biggest killer in Africa at ages less than 15, it may be malaria in some countries. It is a very important public health problem—and there are very few people working in the field.

I learned during this period to carry out basic research in a laboratory and to address very important clinical and basic research questions; reinforcing the

scientific method that I had learned earlier in my undergraduate, graduate, and then post-graduate medical school days.

But now we come back, and you will see my cycle once again. I said, "All right, I have been working with rats for two years. This is a lot of fun, but I need more."

Hannaway: So you go off to the University of Washington at Seattle?

Quinn: So off I go. How I went there is interesting. I decided I had to do a clinical fellowship in infectious diseases, and I interviewed at a couple of places. But one person actively recruited me. That was [Dr.] King Holmes, who is chief of infectious diseases out in Seattle [University of Washington], and who was probably at that time—we are talking about 1979—the leading clinical researcher in the field of sexually transmitted diseases. I decided that he was the person I wanted to work with. He said, "Come out. This is one of the best training programs in the country, and you will do well." He came after me, and I was attracted to him and to the activities that were going on in Seattle, so I went out there.

When I got there, [Dr.] Seymour Clevenoff, who was actually chief of infectious diseases, another winner of the Bristol Award, said, "Why don't you come work with me, because you have done malaria immunology research. We can look at immunologic mechanisms." What he wanted me to do was go back into the laboratory. King was saying, "You came out here to do more clinical training in infectious diseases," and clinical research was what I still wanted to do research on. I said to King, "You are right, and I am staying with you." King was the one who had attracted me there and I owed him a certain obligation. And Seymour and I became very good friends. In fact, King said, "You can spend 20 percent of your time working with Seymour and his colleagues in the laboratory, but the rest of the time I want you working with me."

Something happened in the first week that I was out there during all these negotiations. While I was talking with King—this is a famous story between King and me—he got a phone call, and it was about a patient in the emergency room. King was on call and the patient happened to be a gay man, a homosexual man, who had terrible, very severe diarrhea. They asked King, "What should we culture him for? How should we work him up?" Obviously he got cultures for gonorrhea and asked them to work the patient up for other intestinal parasites. My ears pricked up when I heard this, and I said, "What is going on?" He said, "It is very interesting. There is this big epidemic of parasitic infections among gay men in San Francisco and New York." There are a couple of other people who reported that and deserve the credit. [Dr.] Ben

King, who is now deceased, was one of those people, and there are a few others whose names are escaping me, but I could probably pull them out easily. He said, "Aren't you sort of a parasite expert?" and I said, "Yes." He said, "There is your project. Off you go." He said, "In fact, go to the emergency room right now and work up that guy."

Hannaway: This was in 1979?

Quinn: Yes, 1979. I went over there and I worked this patient up, and he was biopsied. From the biopsy the gastroenterologist on the case diagnosed Crohn's disease. So they started treating him with steroids—the appropriate treatment for Crohn's disease. However, of all the cultures that we had gotten on him, one came up positive. It was for *Chlamydia trachomatis* of a form called lymphogranuloma venereum, or LGV for short. You get it through anal-rectal sex, and what it does is it causes a granuloma inside the colon, and that is a pathognomic finding of Crohn's disease. So a diagnosis of Crohn's disease was being made on pathology that was actually due to an infectious agent. Immediately I said, "This is very interesting. Take him off the steroids, put him on tetracycline." He got better in two weeks, went home, and there were no problems. He did not have Crohn's disease.

Everyone got excited by this. They said, "Tom, you found this. Go and start finding out what other infections these guys get. Go down to the gay bathhouses, go find out what they are doing, what their habits are, whom do they have sex with, how often do they have sex, how much anal-rectal sex," and so forth. At that point, although there had been some studies on gay men, there had not been very many. I did not have much of a literature to work with.

Hannaway: In 1979?

Quinn: In 1979. I decided, "All right, this is an interesting project." In fact, that is what I did for the next...

Harden: You were in Seattle then?

Quinn: This was all Seattle. For two years I worked with King Holmes, and we basically defined the polymicrobial etiology of gastrointestinal infections in gay men. What we found was a Pandora's box. They had everything. They had shigella, salmonella, they had camphylobacter, they had herpes, they had chlamydia, they had gonorrhea, they had syphilis, they had warts--and I could go on and on. They had *Entamoeba histolytica*, *Giardia lamblia*, and other types of parasites. There was everything in there. It was because of the sexual practices that they were engaging in. Large numbers of people were having multiple sex

partners and they did not use condoms. That concept did not exist in the late 1970s. It would not be uncommon to find a man who had had a hundred sex partners in the previous week. This was unbelievable. And it was anonymous sex half the time. That was what was going on in San Francisco and in New York. I was not alone in doing these investigations.

Harden: Yes. Whom were you working with? Were you working with epidemiologists or other scientists?

Quinn: King Holmes has a Ph.D. in epidemiology and an M.D. degree, and he was my tutor and mentor. He taught me the methodology. He assigned a statistician and a microbiologist to work with me, and basically I had a team. I also had a couple of physician's assistants assigned to me. So I was a fellow, yet I had this team of experts working with me. Then I had two junior faculty people also pitching in. One was a man named [Dr.] Larry Corey, whose name will come up later in your AIDS investigations, but Larry Corey was a virologist who had done some herpes work on the anal-rectal area, and he turned all his files over to me because he was moving on to other things. Another was a man named [Dr.] Walter Stamm, who is a famous chlamydiologist, and he helped me with the early description of the chlamydia intestinal infections. By July 1981, I had spent two full years working solely on the clinical epidemiology, microbiology, even to some degree the immunology of these infections in homosexual men.

Harden: But you were not calling this a new disease?

Quinn: No. We were reporting this as epidemics of intestinal infections in gay men, and our early papers reflect that. What are the anal-rectal infections in gay men? Gay men engage in anal-rectal sex, oral-anal sex, and they get contaminated with these fecal organisms, organisms from the intestinal bowel.

When my first report, a sentinel report—although it came out a couple of years later because you know how you have to massage data—came out in *The New England Journal of Medicine*, it was on the chlamydia outbreak in these men. It was followed up by a report on the outbreak of herpes in the anal-rectal area. Then I finally pulled everything together, the whole potpourri of infections, and we put it in as a single paper, "The Polymicrobial Etiology." That was also in the *The New England Journal of Medicine*, and it came out right at the beginning of the AIDS epidemic.

We have not gotten to AIDS yet, but I could be described at that point, having finished all my training, as a person with some immunology training, laboratory training, interest in parasitic tropical diseases, who was now an expert in sexually transmitted diseases among gay men. So that is how I was…

Harden: Poised.

Quinn: Poised, ready for AIDS. I was in the Public Health Service during all this, and then they transferred me. I had finished the training.

Hannaway: You were transferred back to the East Coast?

Quinn: That is right. That is how I ended up back here.

Harden: I want to go over this very carefully. The Public Health Service brought you back here and you were in the Baltimore Marine Hospital. But—let me run through what I know, and then you can flesh it out for me—at some point you hooked up with the Johns Hopkins University.

Quinn: Yes.

Harden: I want you to tell me first about the administrative arrangement for you here at Hopkins, because there are not many people who are paid by the NIH who are working at a university. I would like to know about that. But also, at some point, you start to see these patients as having an immunological problem rather than just having the infections.

Hannaway: Having a range of infections?

Harden: Right. I am wondering if it is because they simply had not gotten to that point when you were seeing them in Seattle.

Quinn: Right.

Harden: Or what was happening? I want you to describe it to me. That is a long question.

Quinn: It is, but that is where I am headed.

Harden: Okay.

Quinn: What happened and how I ended up with Hopkins is an interesting story. When I was finishing up my training, the PHS said, "You have to move." They said, "We have three hospitals you can consider to work in. We need someone at Staten Island, we need someone in Baltimore, and we need someone..." I think it was a place south of Houston.

Harden: Galveston?

Quinn: Galveston. Thank you. I looked at the possibilities. I did not look at Galveston. I was not interested in going down there at the time, nor was my family, so I looked at Staten Island. Being from New York, that made sense. And I looked at Baltimore. The Baltimore Public Health Service Hospital was affiliated with Johns Hopkins. In teaching, patients would go back and forth, residents went back and forth. It was a teaching hospital. So I decided that it would be very nice to be able to be affiliated with Johns Hopkins and be in the PHS hospital.

Interestingly, they did not have a chief of infectious diseases until the end of 1980, the beginning of 1981. That was [Dr.] John Bartlett, who was recruited from Boston, and he became the chief of infectious diseases at Hopkins. So he had just arrived, and he was a single faculty person. He was the division at that time. There was no one else. All of a sudden, here came this new trainee, just finishing training and looking for a position, and I would be paid by the Public Health Service, so it was very nice. He interviewed me, and his interest was in intestinal infections, but it was not the same as mine. It was in *C. difficile*, a different organism, causing antibiotic-associated colitis. But he thought that my intestinal background meshed with his in terms of our research interests, so he said, "I will offer you a faculty position here at Hopkins as assistant professor."

So I joined the PHS hospital, and I became chief of infectious diseases there. Two months later the hospital was sold by the government and it became a private institution. There was a major RIF, reduction in force, by Ronald Reagan, president at the time. I was in that RIF. I was relieved of all my obligations of payback for the training that they had given me if I wanted to, and I could leave the PHS.

However, I got a phone call from [Dr. Kenneth] Ken Sell and [Dr. Richard] Dick Krause at the time, and also from Lou Miller and [Dr. William] Bill Paul. The four of them evidently saw my name on the RIF list. The PHS had said to the NIH and to the CDC—I do not know why this occurred—"If you folks want to pick up one or two people for your individual institutes, you are welcome to do that. These people are all being freed of their obligations, and you can just pick them up." So I got the phone call, and Ken Sell had me down to the NIH with [Dr. Michael] Mike Frank, who also was there, Dick Krause, and so forth. Whether it was the beginning of the AIDS epidemic that made me of some interest to them or whatever, I cannot say because...

Harden: Can you provide the date of this meeting for me?

Quinn: I know it was either in August or September that this was happening.

Hannaway: Of 1981?

Quinn: Of 1981. So the first report was out that there was this thing going on in gay men. They did not come right out and say, "Tom, we want you because you have expertise in working with these gay men." Although the very first time I worked with Dr. Krause, he actually said, "We need more people like you because you have training to address this particular issue. We have to get more people like you. Where are they?" I said, "To be honest, hardly anyone is being trained in sexually transmitted diseases, never mind being trained in diseases that are common among gay men." And he replied, "Well, we need people like that." When I met him, that was the message that got passed along.

When I met with them—that is, Ken Sell, Mike Frank, and so forth—they said, "We're picking you up. We know you from your time working here at NIH on parasitic diseases. Lou Miller gave you a good word, [Dr.] Frank Neva knew you, he gives you a good word. What do you want to do?" I said, "I really want to keep on with my research in sexually transmitted diseases, but I am not sure Bethesda is the best place for that." They said, "What are you thinking?" I said, "I was just getting my research going in Baltimore at the sexually transmitted disease clinics. I could study chlamydia there, and there are gay men that we could follow as well in terms of what is going on with this new little outbreak," which barely had a name at the time. They thought about it and they said, "Yes, but we want you here doing some research." So Mike Frank, who had done this clearance of red cells before, said, "Do you want to get back into that, labeling red cells, but doing it in people this time?" He said, "It might be interesting if you did some of that on these people who are getting this unusual disease, this AIDS." So I was getting tugged a little bit as to what was I going to do at this point, and where.

In between I had a little free time—well, time to make these decisions—and the Fogarty International Center asked if I could help them edit a couple of books that would stem from symposiums over the next three years, one on eradication of measles, one on eradication of polio, and one on eradication of yaws, was it feasible, how far are we from achieving eradication?

Actually, I met some famous people during that period. [Dr. Solomon] Sol Crookman was there; I met [Dr. Samuel] Sam Katz; and I worked very closely with these people in the big battle over the Salk versus the Sabin vaccine. I had to edit their papers. I had to get them to work together. This was all happening during this sort of decision-making process.

But then something happened that led to why I am here at Hopkins, and I will

explain it. Then we will come to AIDS, where I interface there. The NIH at that time had a clinical training program in infectious diseases that was suffering. They did not have enough patients for the fellows to be trained on. To get board-certified, you really need a fairly intensive clinical exposure. I got board-certified in infectious diseases by going out to Seattle and taking care of patients. But the fellows that stayed at the NIH to get their training were not finding enough patients, and the training program was coming into question, which meant loss of board certification for the institute. So Ken Sell, Dick Krause, Mike Frank, and [Dr.] Jack Bennett, who oversaw that, got together and they said, "There is this new person, Tom Quinn, who has this joint appointment at Hopkins. We have occasionally been rotating fellows to Hopkins to get some extra training. He wants to work on STDs in Baltimore. Why don't we set up a situation with Hopkins where we will assign one of our scientists there for a two-year period, and he will set up the training program so that our fellows, whenever they want, can go there and get experience in taking care of patients for a month, two months, three months, whatever they want to do. And Tom can see if he wants to keep his research program going." So they asked me if I would do that, and I said, "Well, I still live in Baltimore, and that would beat this commute every day. I will still come to the NIH once or twice a week and meet with Mike Frank," because I was starting those experiments with him about the clearance of the red cells and so forth inside the gay men with this unusual disease.

At the end of two years, the program was working well here at Hopkins. The NIH was happy with it because all their fellows were getting trained; they got their re-accreditation. I was happy. I was starting to get some good research done. I was still commuting to Bethesda periodically, so I was keeping in touch with everyone. We all decided that we should extend the program, and it has been extended ever since, so here I am.

I am still in the Public Health Service. [Dr.]Tony Fauci is my boss. I was transferred into his laboratory as I did more and more AIDS work. That happened in 1985. I report to him all the time, and I provide an epidemiologic expertise for the basic bench research that they carry out, and we interface on a regular basis on that. As I developed my area and my interest in international AIDS, I was given my own section on international AIDS, although I am not allowed to staff it much with NIH people because I am off campus. There were certain restrictions I had to abide by as Tony and the powers-that-be were not ready with a whole big laboratory, like the Rocky Mountain Laboratory. They did not want another laboratory like the one that was out in Hawaii. The Rocky Mountain Laboratory was already big enough, so they did not want another one in Baltimore at the time. But they could justify having one or two people here, and so they gave me a technician. I have an NIH-funded technician and I have

my own intramural budget. I interface with the extramural people here, and we will get into how that is beneficial further on in the interview.

Harden: I appreciate that.

Quinn: Does that answer that question?

Harden: Yes, it does.

Quinn: What I would like to do now is bring the discussion back to AIDS, what was happening with AIDS during all this, because this is...

Hannaway: Right on the same wavelength.

Harden: We are on the same track.

Quinn: When I came here for the interview—remember I told you Bartlett wanted to know, what is this guy like? You know how it is to go through the interview process.

They presented a case which I will never forget, nor will he. It was at ID [infectious diseases] rounds and it happened to be a woman. This was very interesting. It was a woman who had *Pneumocystis carinii* pneumonia, which was being described at the time in a few gay men. But this was the first time it had been seen in a woman.

Harden: This is August, September, October?

Quinn: This is April of 1981. I was interviewing; I was not here yet. I flew in.

Harden: This was before the first publication?

Quinn: This was before the publication, but word was out that there were a few of these cases. Although that was not discussed too much at the conference. Both John and I remember this very well, because they presented this woman with this pneumonia, and we started saying, "Well, she is probably...." They showed a photograph of her. She was very thin and malnourished, and we started talking about *Pneumocystis* outbreaks in post-World War II orphanages in Europe. That is where *Pneumocystis* was first really epidemic in people. It had always been described in bone marrow transplant patients and those having heavy cancer treatment, but the woman did not have a cancer and was not undergoing chemotherapy. But she had *Pneumocystis*. So we said, "It must be the malnutrition, and she is like these infants [in the orphanages]." We could not

explain it any other way. But when the first report from the CDC came out--which was what, May? Was it May?

Harden: June.

Quinn: It was June. The two of us clicked and said, "Could that woman have the same disease as those men?" We quickly went looking through the hospital records: Were there any other cases? There were no other cases of *Pneumocystis* in people that we could not explain. That was June of 1981. So that was, "All right, who knows what she had," because she subsequently had died in the interim, and she was lost to follow-up. Nothing was left.

Then once more and more was known about this disease, the two of us kept coming back to this case, because we were sure that that woman, who was a prostitute here in Baltimore, probably had gotten AIDS sexually transmitted by someone or it was injecting-drug use. She had come down with it quite suddenly and she did not have the long incubation period that some do. She probably had been recently infected but was a very aggressive, rapid progressor.

But then the CDC report came out. Up to that point, I had not ever suspected that the men that I was working on in Seattle had any major immunodeficiency, nothing like what was being described in New York. I had not seen any of these skin tumors called Kaposi's. I had not seen major pneumonias. The people that I took care of had intestinal infections, and that was all they had. But once I read the reports, I became interested. But I could not study it any more in Seattle, where my big cohort was. I had to move to Baltimore. So I started studying it here right away, and I became, I guess you would say, the first clinician at Hopkins to take care of patients with, I think we started calling it GRID then, gay-related immunodeficiency disease. I started building up a small clinic population of people with this disease and studying them. My first impression, with all the sexual transmission activity that was going on, with these intestinal parasites and other infections, was that it was an overwhelming infection. There was so much infection, it was overwhelming and immunosuppressing the people. I did not think it was a separate, new virus. We are talking about 1981. I thought that their immune systems were overwhelmed by these intestinal infections. That is what I had been studying for two years, so it seemed logical. And I was invited to the first task force meeting on AIDS--that conference that was held at the NIH, not down in Atlanta--which was just being formed.

Harden: The NCI Conference in September 1981?

Quinn: Was that when it was? No. I may not have been there. It was one that I will

never forget, because it was at the NIH and [Dr. James] Jim Curran was there. It was to focus on what the infectious etiology might be.

Harden: So this was after everybody had decided that it was infectious. That was later, then.

Quinn: Yes, that was later. So, it was not the NCI one. I was not involved in that.

Harden: I had to peg it now, but there was a report from the New York Health Department in June of 1982 about hemophiliacs and women and babies, and it suggested blood transmission.

Quinn: Right.

Harden: This is the point from which I date the thinking about infection, but it may not be.

Quinn: Infection. Well, I had been thinking infection, but infection from the intestine that was then wiping out the immune system. My whole training was infectious diseases. So I figured this was an infection that was hitting the immune system. But I was not a virologist, so I did not even really think of pure virus. I just thought this immune deficiency was a secondary effect from the intestinal infections.

I started doing work on that, started studying the patients immunologically, looking at their cell immune response, taking out their T cells and working with that. When flow cytometry came, I quickly went to Ken Sell and said, "I need an instrument; I need to study this," and I have the instrument up here. It has been updated since, obviously. I started studying the patients' flow cytometry, their CD4 cells and how they went down, and their CD8s.

Harden: Now, this was after that conference. Would you talk about that conference and its impact on your thinking?

Quinn: The conference that I attended, at which Jim Curran was present—I do not know if he chaired it or co-chaired it—but he was up at the platform the whole time, always criticizing, always cajoling, always getting us to think. Interestingly, it was not a big meeting. It was a small meeting. Of course, the numbers of infections had not gotten very high; the number of cases had not gotten very high. But a lot of interest in the community was being generated, and so we went down to the NIH. I gave a presentation saying that this disease could be a result of these intestinal infections, and that was about the time my paper was getting accepted for *The New England Journal*. There seemed to be a lot of interest in that, that this was a secondary effect. Other people were saying it as

well as me.

Then individuals—I remember [Dr. Robert] Bob Yarchoan from here got up and said it could be an enterovirus that was doing this. Also, he and I had been looking at blood specimens by EM [electron microscopy], and we saw little viruses in there that we thought were parvoviruses. In fact, they probably were, in retrospect. But we said, "Look at this virus. This is in their blood in a couple of patients, not in people who do not have this disease." So we started looking at the parvovirus.

And I remember Jim Curran. That was my first meeting with Jim. He would get up and say, "All right, come on. Tell me more about this." He would say, "You do not have the right control group, you do not have this, you have to get more patients, you have got to do this." So he was saying, "Here are your negative effects, here are the positive effects, and let us move from there." He did that with just about every presenter that spoke. I respected him right from the very beginning. I thought, "This guy's really quite active." Then he and I got to talking, found out we went to Notre Dame together, and we became friends. I just wanted to get it clear that he and I developed a fairly good working relationship and it has always remained that way.

Harden: He has promised me that we can interview him.

Quinn: Oh, yes. I am sure he will.

Harden: He is the only CDC person that we may have the authority to interview.

Hannaway: Yes, unfortunately.

Quinn: Is that right? In any event, I was working away trying to learn something about the weakness of the immune system, first of all, trying to classify it immunologically, because not many people had done that up to that point. And [Dr. Clifford] Cliff Lane, who was working in our same institute, was working on the B cells. His early paper was that there was a B-cell defect. I was working on macrophages and the reticuloendothelial system and why these people got opportunistic infections. That is where I was doing these clearance studies—they are in the early publications that you will see—and we found that there was a complement defect in these patients.

What we were all trying to do then was to describe the breadth of the immunologic deficiency. I was not really looking for a cause at that point. I did not have that expertise. And although we dabbled in it with those EMs, I was sort of limited at that. I was developing clinical skills on how to recognize the

disease and how to care for the patients and treat their opportunistic infections. So every case that was seen in those early days here at Hopkins was referred to me immediately.

Hannaway: Did you have any more female patients?

Quinn: No. The female was a red herring. It was the first case of the disease that I ever saw, and that is why I brought it up. It impressed me that it was a woman and not a man, because everyone else was talking about men. For the first six months of this, I barely heard a word that any women were getting infected.

But that was all soon to change, because the Haitians were starting to be recognized as a risk group. This was the next phase in my AIDS career: the Haitians getting identified as a risk group and generating a big controversy. I will stop at that point, yes, before we get to Haiti.

Hannaway: We had noticed that you had obviously become knowledgeable about this syndrome early on, and that by 1983, you were presenting a paper at the Infectious Diseases Society meeting, which was held in Wilmington, Delaware. Then the next year the paper was published in the *Delaware Medical Journal*. You were already an authority...

Quinn: Right.

Hannaway: ...on AIDS by that time.

Quinn: With reference to the Delaware issue, I started to get recognized in the region because I was the only one taking care of these patients, and it was because of my familiarity with working with them in Seattle. All of a sudden, I became known as a person who knew how to investigate their diseases and their infections and how best to diagnose and treat them. They were comfortable coming to me because I had a good reputation of working with such infections in Seattle. I was called upon a lot at that point to give talks on what this clinical entity was, so that was probably why I was invited up there.

Harden: Now what I want to do is to shift for a moment, before we continue the chronology, and ask you a question that we have asked many people, just to get your reaction. At this point, let us take it where we are in 1981-82. If AIDS had struck in 1955 instead of when it did, how would the medical community have responded to it? Would they have recognized it? Would they have been able to make any kind of response, and if so, what?

Quinn: I think the spread of this disease among a select segment of the population

immediately draws a certain amount of attention that something is going on. These are homosexual men. Why is this happening? Would it have spread as rapidly in 1955 as 1979? I do not think so. A lot of things socially were changing in the 1970s that were much more hidden in the 1950s. Homosexuality existed, obviously, but not the magnitude of gay bathhouses and promiscuity that was sanctioned, supported, and encouraged in that community in the mid-1970s. That is what made that disease spread like wildfire.

How would the medical community have responded? All of us that are investigative types were going to do the same thing that was done in 1980. We would immediately try to identify how it was spreading, how it was going from one person to the other, just as you would with tuberculosis or you would have with syphilis or gonorrhea. There was a certain amount of medical training that was available to describe the basic epidemiology of this disease.

Immunologically, could we have picked it up? Yes. Those afflicted were developing diseases that were uncharacteristic in normally healthy people, and that would have been identified. Would we have found the infectious cause? No. We did not have the technology or the background knowledge. The building blocks were not yet there for our knowledge of retroviruses and so on.

I do not think we would have identified the virus within two to three years. It would have taken 10 or 15 years. I think we would have struggled for a long time, and we would always have called it a syndrome of unknown etiology the way we call multiple sclerosis a syndrome whose etiology we do not know. For how long did we call peptic ulcers stress-related and not related to *Helicobacter*, until we found *Helicobacter*, and then everything changed. In that instance the bug was found first and then associated with the disease. It would have been a little different, but not the recognition of it.

How it would have been perceived by the public? Probably in the same way. The public was not very supportive of this disease or this segment of the population. There was a lot of outcry and criticism, people saying that this was God's way of saying gays should not be doing this. I can remember articles in the lay press, and many criticisms about the way of life. Even the Haitians were getting the same kind of treatment. Here were these foreigners, they were getting this disease, and what were they doing here? Maybe this was God's way of saying they should not be here. And the hemophiliacs, the Ryan Whites out there and how they were treated and shunned--that would have been the same in 1955 as it was in 1980. I do not think our society was any more receptive in the early 1980s than it would have been in the early 1950s. I just do not think it would have been as big an epidemic in the 1950s. I think there was a real social change from the norm in the free-love format that was occurring in the 1970s in

the gay community. It started earlier in the heterosexual community, but eventually opened up with gays.

Harden: Now we want to turn to the international situation because, in that context, there is much more heterosexual transmission, and so physicians encountered a different picture. I want to return to Haiti. This will be another long question. Let me set it up.

We had a two-hour interview with Dr. Richard Krause, and he described the 10-day trip to Haiti in the spring of 1983, on which you and Cliff Lane accompanied him. He talked about the difficulties that you had getting cooperation from the Haitian physicians in seeing patients. He noted that the first three patients you saw were women and that three or four of the first 10 had tuberculosis. The two-part question is: Was this the first time, or was there some other time, when you realized that AIDS was an international problem? Then would you begin to talk about the Haiti trip.

Quinn: There were reports coming out of Europe of this syndrome, and we knew the Haitians were getting it. It was pretty obvious to me that if Haitians were getting it, they had to be getting it in Haiti as well as in New York and other places in the U.S. But how did the Europeans link into it? With gays going back and forth, maybe it was transmitted that way; the shipment of blood products and things like that, because we are now talking about 1982 and 1983, and the recognition of it in the blood supply was occurring around that time as well.

But then it was Dick [Krause] who called me and said, "We have been asked to go to Haiti." I guess the Haitians first came up to the NIH, to the Fogarty [International Center], is my recollection.

Harden: I believe so.

Quinn: And the Haitians objected to this. And the CDC came to the meeting. The NIH was there. I do not know who sponsored it. The Haitians came and they basically said, "We do not want the CDC. They are the ones who have labeled us. But we will allow a team of investigators from the NIH to come down and invite them to find out what is going on." However, the CDC said, "We have to have at least one person on the team." It was, I think, either [Dr. Albert] Al Saah or [Dr.] Harry Haverkos, or maybe both. Both of them were at the CDC at the time. Neither is there now, but back then they may have been part of the AIDS Task Force. So Dick accepted the invitation to go to Haiti. I think PAHO [Pan American Health Organization] was somehow involved. Dick asked me to join him.

I was being asked, I think, because I had the most clinical experience of anyone in the NIH group. Yes, [Dr. Anthony S.] Fauci and Cliff Lane and others had seen patients, but I had quite a big practice by that time. Not that I intended to. It was just that Hopkins was a nice place to go if you had this disease. So I had seen quite a bit. I was in the thick of it in terms of the investigation, so it made sense that Cliff and Dick and I, as members of the one institute, go to Haiti. But I vaguely remember that there was either Harry Haverkos and Al Saah who I think were both CDC [personnel]. That was the team that went down.

Dick is absolutely right. The first thing that we saw were these women, who were just wasted away, coughing, probably having *Pneumocystis* or tuberculosis or whatever, and we were told that they had tuberculosis. They showed us the X-rays. I never actually saw definitive proof of that, but it was suspected. I think Dick is absolutely right on that account. But, boom, it hit me that there was a comparison with the first woman with the disease that I had seen.

It brought home to me that, number one, this disease was not affecting just one gender; it was probably going to hit both. It looked like it was still sexually transmitted because the woman's husband was usually also sick or had died, so I could link it back to that. We asked them lots of questions: Why were these women getting the disease if it was only supposed to be in gay men? Yet there were millions of other Haitians, who were not infected, who were not showing signs of the disease. Again, we did not know what the cause was at this point. So, as we were sitting back at the hotel, we talked about how this really looks like a sexually transmitted organism or an organism that heterosexuals are getting and homosexuals are getting that somehow gets into the blood supply through blood donation. Now, we were not talking about needles at that point, even though dirty needles are used in Haiti for medicinal purposes. But I think as we went around those clinics, it was clear to us that there was some evidence of heterosexual spread. Although we did not set up an investigation to pursue that, we laid the groundwork for future studies to develop and to investigate that.

I met [Dr.] Jean Pape, who was affiliated with Cornell and was doing studies there; [Dr.] Warren Johnson, who was his mentor; and [Dr.] Neil Halsey, who was doing a measles study in Haiti. So right in those early times I started to meet the other people, the Americans, who were working in Haiti on other related diseases. Jean Pape and Warren knew of my work of intestinal infections right away, and so we headed off and were starting to exchange information.

Then I returned to the U.S. with this impression in my mind that, yes, this

disease was prevalent in Haiti. The way it was described was that gay men went to Haiti for vacations, and they went to these poor Haitians, who would do anything for some money, and would engage in homosexual acts even though the Haitian men might be heterosexual. Then the Haitian men would go back to their wives and infect them. The scenario, as I recall it, that the popular press and others got at the end was that this was still not "really a heterosexual disease." Women were not spreading it to men. This was solely male to male and male to bisexual male, if you will, who then gave it to the woman. But the woman never gives it to the man. No one in 1983 thought that could happen that I can recall.

Harden: In the United States?

Quinn: In the United States or in Europe. After being in Haiti, we thought it was very possible, so we were calling it a heterosexual disease and maybe one that went both ways, because we saw equal numbers of men and women. Eventually, the longer we stayed down there, the more equally divided were the numbers of patients, male and female. We were saying, "This can't all be going that way." But that is what the lay press tended to think.

Then there was a report in Europe of Africans with the same disease who had come from Zaire and other places to Belgium, and France, and the patients were both men and women. That was all I needed to see. It was, I think, just one report, but that was enough for me. I felt this was not just a gay disease, and I doubted that this was dirty needles. But the only way we were ever going to find out was to go to Africa or go back to Haiti and set up good prospective long-term epidemiologic studies.

This was at the same time that people were talking about doing that anyway with gays, here in the U.S. and in Europe, that is setting up prospective cohorts and finding out how this was spread, what was causing it, and so forth. I think 1983 was a very instrumental year, early 1983, for setting up cohorts to investigate the epidemiologic as well as the clinical natural history of this disease.

Then I guess Dr. Krause probably filled you in, but this is an interesting story. [Dr.] Peter Piot had trained in Seattle, and our paths crossed. We met there, but we did not really share any particular research. But I had met him, he had met me, so we knew each other. I know a few other things from reading Laurie Garrett's and other people's books, as to what might have happened, but I was not privy to that. When he came to me—this is the way I remember it—everyone has their slightly different stories, but they are all very close.

My recollection is that Piot and I were talking about this spread of the disease, and that I had been in Haiti. I told him what I had seen, he told me what he was seeing in Belgium, and we were saying, "We really should set up a project in Africa." He said, "I have been trying to do that. I can't get funding for it. Do you know a way that I can?" I said, "I work for this man, Dr. Krause, who is interested in setting up projects overseas to help internationally to try and figure out what is going on. We were just in Haiti together and we talked about Africa, because many of these Haitians had lived in Africa." I said, "Do you want to meet him?" He said, "Sure. If he'll fund a trip, that would be great." So I introduced Piot to Dick. Now, whether he went to Dick first and then they brought me in, I cannot remember—my recollection is that I introduced him to Dick, but it could have been the other way round. It was a long time ago. But it seems that Garrett thought that it went that way. Piot went to Dick and then Dick called me like he had called me for Haiti. Whatever. I do remember that we sat at a sidewalk cafe. Has Dick told you about this one?

Harden: I do not think so.

Quinn: It is really interesting. I was talking to Peter, and Dick was walking by with [Dr.] Jack Whitescarver, and we pulled them aside. We sat down and we planned the project to go into Zaire, to Kinshasa, and find out what was going on. [Dr.] Karl Western got involved in that. It was planned that we would all go to Antwerp first, meet with Peter in the Institute of Tropical Medicine, since I and my colleague, who had been appointed—[Dr. Frederick] Fred Feinsod, I think it was—was to join me. He was also new with the institute. We would get educated about what life was like in Zaire—that it was the former Belgian Congo, We got a real history education—what the diseases were there, and things like that. Then we would fly down to Kinshasa two days later.

Dick set up the funding for it; Ken Sell set aside some funding for equipment. I was to go as the clinician because I had seen all these AIDS cases in Haiti and in the U.S. Piot would go as the epidemiologist and as the person who had previously been in Zaire. Feinsod was another epidemiologist along to help with some of that work. There was a man named [Dr.] Henri Talman, who was also a clinician and who had seen a couple of Africans in Europe, and he was going to join me. So we had this team to go to Zaire.

Then, before we left, we got a phone call from HHS [Health and Human Services], [Secretary Edward] Brandt, saying, "We have your foreign travel notification in the office here. It is approved. But we have another one from the CDC—they are going to Zaire—from a man named Joseph McCormick. Do you know him?" "No, I do not know him." "I do not want two agencies, the CDC and the NIH, tripping over each other. This has to be a joint thing. Please

call him up and invite him." I said, "Okay." So I called Joe up and I said, "I know you are going. We are going. Why don't we go together. Why don't you join us in Antwerp." He said, "Fine. Who are you working with?" I said, "Peter Piot. He's the organizer." He said, "Peter? I worked with him a long time ago. We worked on Lhasa fever and on Ebola fever. I know him fine." "Okay, you guys know each other." In fact, they did. They had worked together previously, in 1976, I think it was. So McCormick joined us and we flew to Zaire together.

We got there, went to the main hospital, Mama Yemo Hospital, and interviewed the chief of medicine there. He showed us all his AIDS cases. We went on rounds and I kept saying, "AIDS, AIDS, AIDS, AIDS." The cases were just lined up. It overwhelmed what I had seen in Haiti. It was far, far more devastating, with people just wasting away completely.

I had brought some laboratory equipment along, and we tried to set it up. We had some problems with the monoclonal antibodies because African blood types are different than American blood types, and so no one had any CD4 cells, initially, because the monoclonals were not picking them up. That got corrected eventually. The patients did have CD4 cells, and the cells were not zero.

Hannaway: Was that what you thought initially?

Quinn: At first we were thinking, "My gosh." But even the healthy people had no CD4 cells, so we knew we had a reagent problem. But that got taken care of. We stored all the blood, and we tied it together. Peter and I and Joe became the leaders of the team, each one of us taking on a different responsibility.

Hannaway: How was that division organized?

Quinn: Joe had never seen a case of AIDS before, nor had Peter had much experience, so I clearly was the "AIDS person." Peter, again, was the Belgian-Zairian connection, playing those politics; and Joe was the American-Zairian politics, which are very important in doing a foreign investigation—very, very important. I stayed out of the politics part because I did not even know how to speak French. I was in the dark whereas those two were negotiating left and right through the Belgians and through the Americans and the Zairians to allow us to do our work, because Africans did not want to be labeled as a risk group like Haitians, which was what was happening in Europe. There was a lot of sensitivity.

So the two of them handled the politics. Joe was good at setting up the repository and helping me with the laboratory part, whereas Peter was very good at interviewing different doctors down there and finding out what had they been

seeing, and trying to track how many cases there had been in the past. Because, from my perspective, Peter was the original instigator of this investigation, and, as we built our clinical description of what we were seeing and how many cases we were seeing and so forth, I think it was pretty unanimous among the three of us that Peter should write the first paper and that it should be in the *Lancet*. He did that, and that was our first paper on the epidemic in Africa.

Hannaway: Laurie Garrett claims that the team that visited Zaire in 1983 had difficulty in getting a paper published in the *Lancet*. It was only after rejection by a number of other journals that it was published in the *Lancet*. She claims specifically *The New England Journal of Medicine* rejected it, but she also mentions another journal. We wondered if this was accurate, and, if so, why do you think the paper was rejected? But from what you are saying, it does not sound as though the paper was rejected.

Quinn: We had decided it should go to *Lancet*. We might have said *The New England Journal*. But my understanding is it got published fairly quickly.

Hannaway: You were in Zaire in September 1983 and then this paper was published in July 1984?

Quinn: It came out, yes. So it was published in July. We were there. But the paper did not get written until probably close to January, because of all the tabulation and data. Normally, it is a six-month delay anyway with one journal, never mind going to three journals. *The New England Journal* takes two months for review at the least. It might have gone to *The New England Journal*, but again, I think the only person who could answer that is Piot, if you ever get to interview him. He will tell you.

Hannaway: All right. We will try and add that to our list.

Quinn: Yes.

Hannaway: Let us come back to this trip to Zaire. What were the chief differences clinically of the Zairian patients that you saw in the Kinshasa hospitals compared to the American patients, and also the Haitian patients, if you like?

Quinn: I have to say what the similarities were first. Since I was already oriented towards the intestine and the gut, what I saw was diarrhea wasting syndromes in both fulminant diarrhea and so on. That was why I was still focused on it as some sort of unusual intestinal parasite or organism that was causing this overwhelming diarrhea. And they called it "Slims" disease. That is what it was called at the time in Africa. They did not know what was causing it, and they

did not call it AIDS. It was called "Slims" disease.

Hannaway: They called it that in Zaire? I thought that was primarily in Kenya.

Quinn: It was. They had some French term for it, but they also called it "Slims" disease. I do not know what the French translation was at this point. But Peter was my translator, so he would say, "They're saying "Slims"disease."

The patients in Zaire had an unusual rash that was different, and we did not know how to describe it. We took a bunch of skin biopsies, gave them to dermatologists, and it was nonspecific. No one has ever really figured out what it was due to. Some say it was insect bites that the patients got super-reactions to.

But I did not find that the patients in Zaire had different symptoms. In fact, I was looking for similarities, not differences. The only difference was the appearance of both equal numbers of men and women as patients.

Hannaway: Did you see Kaposi's sarcoma?

Quinn: Yes. We saw the classic Kaposi's. I saw the same type that we saw in the U.S., and it was in the intestine as well as on the skin surface. My impression was it was the same disease. I had no doubts in my mind. What I saw in Haiti, I was seeing in Africa, and I was seeing in the U.S. The color of the skin might be a little different in that I tended to see more white men in my practice here at Hopkins, and I was seeing black Haitians and black Africans. But other than that, and seeing more women, it was essentially the same.

Hannaway: You were already, in a sense, inclined to accept that there was a heterosexual transmission?

Quinn: Yes, I was. Even though I had worked solely with gay men and gay transmitted diseases for two and a half years in Seattle, and then coming to Baltimore, and then recognizing this disease, seeing these equal numbers. How were these women getting it? Then, we heard about the prostitution that goes on and the multiple sexually transmitted diseases. As far as I was concerned, after my trip to Haiti, I was convinced this disease was heterosexually transmitted. In fact, it left such a mark on me that when I came back from Africa, although I was still involved with the African project and getting it to be a much longer prospective cohort study, the first thing I did was I said, "We have got to start looking for this in our clinics at Hopkins and looking at women." I said, "Let's start saving blood away," and you will see where that comes in, because that was the source of the real battles with the press that I had at the time.

Hannaway: Please continue.

Quinn: All right. Well, this skips a couple of years, so I did not want to jump to that yet. But let us just leave that as "see footnote."

Harden: I wanted to have a few more specifics on Projet SIDA as a project in terms of how it was developed, the personnel who were involved, the goals, how successful it was by the time it was shut down.

Quinn: But first, before I do that, because you have heard all about the early development of my career, what I wanted to say was, when I was asked to go to Haiti and to Africa, I could not have been more excited in the sense that that was what I had trained for, to do international tropical medicine, and here I was going into the tropics. In fact, one of the reasons why I left the Laboratory of Parasitic Diseases at the NIH was that I did not see any career from there going off into Africa to study malaria. It was all laboratory and mice. Maybe it would have eventually led to that. But when Dick asked me to go to Africa, I was, "Wow, this would be great!" This was what I, from my early days in undergraduate school, wanted to do. I said to myself that I would continue the international part as well as setting up a domestic research program of my own.

But now to approach Projet SIDA. I left [Zaire] early, actually. I flew directly from Zaire to Aarhus, Denmark. Aarhus was having an AIDS meeting and they asked me to report. So I left Peter and Joe and the rest of the team down in Zaire, and they continued doing their work. I was the first one to leave. They said I could go ahead and give a preliminary report, and I told the people in Denmark what I had been seeing. Everyone got all excited. There was no credibility problem that we were seeing what we thought was a heterosexually transmitted disease or a disease that affected both men and women.

Then I flew from Denmark to Las Vegas to the infectious disease meetings and again gave a very preliminary report on the findings of Projet SIDA. That seemed to be fairly exciting. None of it was published, so it was not in any literature yet. But I did not have any negative feedback.

Joe and Peter had finished by the time I finished going to these meetings, and we got on a conference call and we said, "We have got to do something. We have got, one, to write the paper up, so we are talking like November; and, two, we have got to set up a prospective program." And it was the three of us. For some reason, I do not know why, I guess because we wanted one person to represent each of the institutes, the Institute of Tropical Medicine, the National Institutes of Health, and the CDC. So it was Joe, me, and Peter, and in reverse

order for each of those institutes. We each said, "Okay, here's how we have to do it. We will go to our respective people, find out who can get the most money, set the thing up, and we each assign a person." That was how it was left.

About a month later, there was a new conference call. Joe was no longer on the team. He was out. Jim Curran was in. I cannot tell you the politics that went on. I do not know. I was not privy to that. All I know is that Jim said, "This is within our purview; this is an AIDS-related issue. I am in charge of the task force. I agree with Joe that there should be a team there." He said, "I have already identified someone whom I want to send there. His name is Jonathan Mann." I said, "Send me his CV so I can see what he's done." And Peter said the same thing.

I identified an American to go by the name of Skip Francis or [Dr.] Henry Francis, who had been working in the Laboratory of Parasitic Diseases, seemed to be very knowledgeable, and who had worked in Africa once before. I wanted someone who knew what he was getting into, and whose task was to set up the immunology part of the project. The CDC was to set up the epidemiology part. Peter Piot was to set up the clinical part, and he identified a man named [Dr. Robert] Colebunders. But Peter said, "Tom, you have more clinical experience. Can I send him over and you train him?" and so Colebunders came over and trained with me, and Skip was transferred to me. So I trained the two of them as to what these patients looked like, what we should be doing, and we designed research protocols and so on like that.

I got—this is a funny story—the CV of this Jonathan Mann, who was a health officer from the CDC in New Mexico studying, I think, it was rabies at the time. Wasn't it rabies?

Harden: Amongst several things he did.

Quinn: I said, "He does not have any AIDS experience. Why is Jim doing this?" So I said that to Jim Curran, and Jim said, "He is great. He's a very good epidemiologist. He'll do a good job." So I said, "Fine. He's your selection. Here's my selection. Here's Peter's selection." And off it went.

So it was decided that they should start. It was, as I recall, around June or July of 1984, the same time that the paper came out, that the three of them went over. Jonathan got there first, then Skip came, and Colebunders came. Colebunders was a very nice gentleman. I really liked him. I had not met Jonathan Mann yet.

Because we were going to be responsible for the laboratory, Skip went to Ken Sell and they bought all this equipment, loaded up some C131 Air Force jet or something, and we flew it over. It was a huge amount of equipment. Ken Sell was great that way. He thought big, and you needed something big to take on these investigations.

This was how it was set up. The NIH part was to do the laboratory work—diagnostics, the immunology. The CDC was to do the epidemiology: describe if the disease was heterosexual, if it was perinatal, how big a problem it was, and so forth. Colebunders was to describe what was this diarrhea wasting, what was this SLIMS disease, what were the characteristics, the similarities and dissimilarities.

I would not say that they all got along very well, but I would say that in two years, that team generated all of the information that led to our understanding of the AIDS epidemic in Africa. It was those three gentlemen, with Peter, myself, and Jim Curran as their back-ups, their directors, their mentors instigating them, do this, do that. None of that would have been possible had it not been for the discovery of a virus that was causing AIDS. It would have been very difficult. We would have been stumbling along in the dark: who was infected, who was not, who has got what?

That brings back a funny political story as well. Here I was sitting on all this African sera from the first investigation [in Zaire], and I started getting tugged in two ways: should I give it to [Dr. Robert] Gallo to go test or give it to [Dr. Luc] Montagnier to go test. I did meet with Bob and we talked about it. But Peter was good friends with Luc Montagnier. And I was at NIAID, and it turned out that Martin...

Harden: [Dr. Malcolm] Mal Martin.

Quinn: Mal Martin was good friends with Montagnier. They said, "Send the sera to Montagnier." He had that first paper out on LAV, and so we did that. And the ones we would have called AIDS were all positive, but a fairly high percent of the controls that we had picked were also positive. So we thought, "This test needs some work." In retrospect, they probably were all infected but had not developed the disease yet, because we had picked relatives, and, in some cases, they were wives or husbands or spouses. But that was from the first investigation.

We published that in *Science*, by the way, and there was no problem getting it published. I was last author on that one, and one of the French investigators was first.

Hannaway: What date was that published in *Science*. Was that 1984 or 1985?

Quinn: Which year? I think that was 1985.

Hannaway: Yes.

Quinn: Or it may be 1984. Again, let us find out.

Hannaway: Okay. It was 1984.

Quinn: It was 1984, at least that is what I have here. Brun Vesenet is the first author, Montagnier second, and Barré Sinoussi is on it. Then it is McCormick, Piot, Talman, and a number of other people, and then myself as last author. That was in *Science*, 1984. And I have here volume 226, pages 453-457. So that is the reference. If I am off, it could have been the end of 1984, but that is about where it was.

Harden: You have that gap. The virus publications were in 1984 too.

Quinn: Eighty-four as well, and this was his test, that Brun Vesenet was the one trying to make the serologic test. We sent the sera, picked up all the ones we called AIDS, but there were others that were still positive, so they were worried about specificity with that.

Then Abbott came out with their first test in *Organon Technika*, and I went to them before these commercial outfits were licensed, which were using the Gallo test. I actually got it sent to my laboratory. I did not have to send the sera to Gallo at that point because it had now gotten into the commercial outfits. I tested the same sera, again with almost identical results. But at that point, we realized, because of the papers that were starting to come out, that these people were infected but did not have full manifestations of the disease yet. The way we did that was their CD4 counts were much lower than in the people who were seronegative. We used the CD4 count as an aid to help the specificity. But once these tests were available and we had published our African experience, as the laboratory overseer in the project, I immediately said, "Let's get these kits over to Africa." We were the first one to get kits into Africa actually to do blood testing.

Jonathan Mann, who was a quick thinker and very aggressive in his epidemiologic approach, said, "Let's go study this population, and this population, and this population. Let's use those serologic tests and let's find out what we have got going." If you look, in terms of the publications, he was

publishing like every three months. You may say, "How was that possible?" It was possible because in Projet SIDA, even though the individuals had some personality quirks with one another—you will hear plenty about that—they did good science and they worked hard together. Maybe they worked individually, but eventually they had to put the data together. And maybe it was the six of us that made that possible.

In other words, Piot and myself and Curran got along very well with each other, and we got along with our respective individuals. I can remember meetings when Jim would fly up here, Jon Mann and Skip, who were not talking to each other at the time, would fly here, and myself, and the four of us would negotiate how to get out of a particular wrinkle in an investigation. Whether it was political or science or whatever, we would work together and we would end the meeting with resolutions, and out would come a paper two weeks later.

So it worked. Sometimes opposite personalities can be complementary, whether they realize it or not, and be creative, thinking in different ways. Plus my laboratory then pitched in. I said, "If you're not getting the work done, if Skip can't get the work done quickly enough, I will start having my laboratory back you up," and so forth. The phone calls were intermittent, but I was on the phone with Jonathan Mann or Skip almost weekly at that time.

The clinical studies were a little slower to get going. They took longer because you needed a lot more patients to say, "All right, we screened 200 patients," with the disease, "and here are their characteristics." I do not remember as much heated activity going on [in that part] as I did with the first part.

The project itself was called Projet SIDA. It was endorsed by the U.S. Embassy, and got its blessings. Jonathan was a very good politician, as you will hear later on. But he got the American Embassy to adopt this project under its wing, and give it lots of support. The NIH and the CDC split the money to get it going, and Peter got money out of the European Economic Community. So it was jointly funded by all three parties. I was very excited about it. It was a real highlight of my scientific career to be able to work with other institutions overseas and for us all to work together towards a common goal, which was to learn more about the spread of this disease.

For me, the crowning event in the early days of those first two years was the piece that I was then asked to write, and that was "The AIDS Epidemic in Africa: Epidemiologic Paradigm."

Hannaway: That appeared in *Science*.

Quinn: Which was in *Science*.

Hannaway: In 1986.

Quinn: What I did was I sat down with Jonathan and Skip and Jim and Peter and I said, "Let's pull all our data together. What are we saying here in all these individual little pieces?" Because what was happening was that Jonathan would do a little study and, boom, he would publish it, and then do another. I said, "Let's put it together and let's summarize it. Let's make it a package and let's put some new data in to make it even more exciting." That was that *Science* piece, and I am very pleased that they all supported me in that effort. We got along very well, in terms of getting that done.

I know there was all this friction in discussions, the friction of one group versus another. Yes, the CDC and the NIH were a friction waiting to happen. If you take an individual from each of them and say, "I want you to represent our institution and go in there and do your thing, and you represent your institution," they will have the same kind of frictions that were happening back here in the States. So I do not think it was unusual. Plus you get pretty isolated out there. The members of the team have actually remained friends, and I just saw them all in Vancouver. We happened all to be passing through at the same time, and we all stopped, and it was really exciting to see the whole project group back together. So that was the first two years of Projet SIDA.

Then Jonathan, who was clearly a rising star, got recognized by WHO [World Health Organization], and off he went. He was replaced by [Dr.] Robin Ryder. Robin was a different kind of person. He was a CDC person who also had diarrheal epidemiology training, but, again, his role in Projet SIDA was to oversee the epidemiologic aspects. He thought differently from Jonathan. Robin believed in bigger cohorts followed for a longer period of time resulting in a much more important finding, whereas Jonathan, at the time, was interested in who was affected and how quickly were they affected. He did not want to define the natural history. His aim was to say, who was getting it here, and why were they getting it? Whereas Robin said, "I want to know, once you are infected, who do you transmit it to eventually, and how long can you stay alive or how long does it take for you to die from that disease?" So he set up these huge cohorts and followed them. His most famous cohort was the pregnant women who were HIV-infected. He described—I think, probably the best paper was in *The New England Journal of Medicine*—the perinatal transmission of HIV in Africa, what the percent infection rate was, the clinical outcomes in the infants, and did they take to vaccines or not. There were a lot of spinoffs. So the real natural history of HIV in Africa—not its epidemiology, how it spread, but its natural history—was being dissected by Robin Ryder.

By that time, Skip had been in Zaire three or four years and it was time for him to come back. I put in another person named [Dr. Christopher] Chris Brown. Peter Piot had been publishing some papers from the project on the clinical aspects of Bob Colebunder's work, and he left and was replaced with a man named [Dr.] Yost Perriens. All these people published their own papers. Perriens got into tuberculosis and HIV, which was very interesting.

Those investigations continued for a couple of years doing fine, with, again, the usual frictions. I would say those frictions between the individuals sometimes were due to personality, but were sometimes driven by institutions. We had our own needs, and sometimes those needs stepped on someone else's needs and vice versa. Eventually I pick up the phone, call and talk to Peter or talk to Jim, and we would solve it. There was not usually a problem. They called us the "patrones," French for the overseers, I guess. And we would go there once or twice a year, spend a week or two weeks, do a scientific review and so on. Then things started getting heated up politically in Zaire.

Hannaway: In the late 1980s?

Quinn: Now we are talking about the early 1990s. And the studies were still ongoing. It was getting more expensive because of those huge cohorts that Robin set up. I will never forgive Robin for that. I admired him for his scientific epidemiologic expertise, but, meanwhile, the project was going bankrupt because he had a lot of cohorts going on, and they are expensive. We went from a small project of maybe 25 Africans to having 300 Africans, Zairians, working in it. It was huge. We went from two cars to 12 cars and trucks. This became a big project with I do not know how many freezers, perhaps 25 freezers of specimens.

I should mention that the Armed Forces Institute of Pathology joined us midway through the tenure of Projet SIDA, and they did some pathologic investigations as well.

But then the politics of Zaire became unstable and there was rioting in the streets. We were worried about our people, and actually they were airlifted out and have not returned since.

Hannaway: This was in 1991?

Quinn: 1991. Although some of the Africans that we left stayed with the project and have persisted, Projet SIDA is not producing anywhere near what the original project was when it had all that support. It was publishing 10 papers a year in

major journals and really writing the history of AIDS in Africa.

There was one other project that rivaled us, I would say, and that was the Nairobi project, which Piot and I were also affiliated with. The CDC was not. Eventually, even I was removed from that. I removed myself because I got interested in the Orient. That is another story. I am looking at my map. The CDC set up a separate project independent of the NIH to get away from some of that friction, but also in an important area, West Africa, to look at this HIV-2, and that was in the Ivory Coast. Piot, for the clinical side, was still affiliated with that. So Piot was involved with all three of those major African cohorts.

Then Kampala, Uganda, eventually got a project going with Case Western and a couple of other universities. In fact, I am now in Kampala with a project in what is called the RAKAI project, which is operated out of Hopkins and Columbia [University].

Harden: What happened to the 25 freezers when everybody left? Did the specimens come back to the United States?

Quinn: The situation was that we split the specimens with the Zairians. We could not take them all, but separate aliquots were made, and we took half and they kept half. Ours are in Rockville, Maryland [at the American Type Culture Collection].

Harden: Are they still available?

Quinn: Still available, still there. All you have to do is come up with a research idea.. We just investigated the role of vitamin A in pregnancy and perinatal transmission. That study is just completed. We did another one on PCR [polymerase chain reaction] and viral load. We just pulled the specimens out and applied our tests. Because the epidemiological databases are still there. They are there at the CDC, and I have the specimens, and so we just get together.

The person who oversees that—as we got into the early 1990s, people on the team changed a bit, and a new epidemiologist was assigned to it—a man named [Dr. Michael] Mike St. Louis, who was just great. He was not the lead CDC person. That was still Robin Ryder at the time. He was a junior epidemiologist, but he was very, very good. Mike is now in sexually transmitted diseases at the CDC. He was no longer associated with AIDS when this whole thing came crashing down.

Some people on the team said, "I have had it." My investigator ended up as a

physician in Wyoming, not doing any more research.

Harden: Was this Chris Brown?

Quinn: Yes, Chris Brown. Yost Perriens ended up at WHO; he is still working there. Robin Ryder ended up at Yale. He got a professor chairmanship there. Jim Curran, as you know, moved on to Emory. Other people are moving on and I am still doing the same old thing!

I enjoy what I do. I enjoy this dual relationship between the NIH and Hopkins and being able to do international research. And we have moved on from doing the Zairian project, into Uganda, into Tanzania, and India, Malaysia, a little in Thailand, and a few other places.

Harden: What I wanted you to do to finish off today's interview was simply to compare Projet SIDA with the Multicenter AIDS Cohort Study [MACS]. Were those working in this country aiming at the same things, and was the MACS as useful as Projet SIDA or more or less useful?

Quinn: No. They had different aims. Projet SIDA was quite diverse in its origins in the sense that it was not necessarily designed to describe the natural history of AIDS in a population. Actually, that was what the MACS was designed to do. [Dr.] Frank Polk was one of the big instigators of the MACS. He was here at Hopkins, and, in fact, we worked closely together. They really wanted to enroll people who were HIV-positive and find out how long it took for them to get sick and what could be done to prevent that. It was a real natural history of the disease project.

Projet SIDA, when it was developed, was how is this disease being transmitted? What is causing it? Is it the same virus? How was it being transmitted? That was not an aim of the MACS, how was it being transmitted. And then, in Africans, is the clinical disease very different? We really did not have the natural history aim. Yes, we listed it as an aim, but we knew that in the short term that was not going to be answered. For that, you needed a much longer-term, stable project of a cohort.

It was not until Robin Ryder showed up on the scene that he started to set up these cohorts, of commercial sex workers, of prostitutes, of pregnant women and their babies, and also of workers in businesses. It was very interesting how he did that. Robin had the same ideas as those organizing the MACS, but organizing the cohorts was not until 1988 or 1989, and that only lasted two and a half years to three years, and then it was shut down. MACS is more than 10 years now, so Projet SIDA did not even come close to MACS.

Is it as valuable as MACS? Yes. I think AIDS is an international disease that goes well beyond our boundaries. We have learned not more, but different, types of information from the African project. We learned that there were different viruses there, the different genotypes. That information came from our original investigations, that the Zairian strains were different from the American strains. We found that heterosexual transmission was occurring. We defined the rate of perinatal transmission, the risk factors for transmission in a developing country setting, where 90 percent of all HIV infection was going to occur in the future from here on in. And I could go on about different clinical features and so forth.

We do not have the treatment to give these people in Africa that we do in the States or in Europe. How do you take O76, AZT, to prevent transmission in pregnancy? In the United States, you give it for six weeks to the pregnant woman, six weeks to the baby. You give it intravenously during birth. You cannot do that in a developing country. You are lucky if you get a dose or two in the woman when she is in labor and maybe a dose or two in the baby before they leave the hospital. Then you have to see whether some shorter course is going to work. And those studies are underway.

That was the aim of Projet SIDA. It was to take the beginnings of the knowledge of this epidemic in the United States and Europe, what we did know, and ask the same questions in Zaire: Is this the same disease? Is it spread the same way? If it is not, then why is it different? And if it is different, will it become different in the United States?

That is where we then come back to heterosexual spread, because it was after our 1984 paper and our subsequent studies in Zaire that I came back to Baltimore and said, "This disease is heterosexually spread. It has to be." I started looking at the saved samples I told you about with the new diagnostic test. And—this was published in the *Journal of Infectious Diseases*—what we found was that a high percent of the infected cases attending our STD clinics were all men, from 1978, I think, to about 1983. All of a sudden, women started showing up. Their numbers started increasing till they almost reached a one-to-one sexually transmitted rate of infection. So then we said, "How are these women getting it here? If it is like in Africa, it is heterosexual transmission, I bet." But injecting-drug use turned out to be a big co-factor in the spread of HIV in these women. But many women got it because of sexual exposure to a man with known risk factors, and because she had syphilis or herpes or a sexually transmitted disease, just like what we were finding in Nairobi and in Africa, in Zaire.

That is when I got into my problems with the press, because it was in 1986 that I started talking about this actively, saying, "What is happening in Africa is going to happen here in the United States." People said, "You do not have any proof, any evidence." By 1988, I had all the evidence I wanted. I published it the information in *The New England Journal of Medicine* and then I got attacked left and right. Critics said that these people were lying, they were really having sex with gay men, and they were shooting up and just denying they were shooting up. So my paper about heterosexual spread in STD clinics in the United States--it was in Baltimore, but it is U.S.-based study—came out about the same time that *Cosmopolitan* came out with their front cover saying, "Women, you do not need to worry. You can't get AIDS." I started going on these talk shows and television interviews and the interviewers said, "You are just an AIDS investigator who is trying to get money to do your research, and so you are saying it is heterosexually spread." I was replying, "No, I am not saying this for funding purposes. That would be insane. I do not need to because my funding is for work in Africa anyway." It was not until a couple of years later that other papers and reports, other scientists, were starting to support the idea that heterosexual transmission might really be happening here in this country. But it took a long time, I think, for us to come to that realization.

Harden: That is very interesting in a variety of ways, in part because you were a scientist, you were working from data. The data had convinced you. Why did the press, the media, or the talk show people, why were they not as convinced by data as you were? What were their objections?

Quinn: They were looking at AIDS cases. Counting the AIDS cases—not those with HIV.

Harden: Is this from their lack of scientific training?

Quinn: I will never understand it.

Harden: This comes up in a variety of medical situations.

Quinn: Yes, I know. I cannot understand why. I do not understand why. I will give you one example. There was a reporter here in Baltimore who writes a column every day, and he saw my report in *The New England Journal of Medicine* and an article about it in the *New York Times*. The *New York Times* was very supportive because its article was by Larry Altman. Larry Altman is a well-trained person, and he saw the data and was convinced. He said, "Okay, so some of the women may be lying, but not all of them. They are getting AIDS sexually. They have to be."

Well, this Baltimore reporter did not call me. He called up the state health department. He said, "Look, there is this report about HIV infection. What are the statistics?" The state said, "We do not collect HIV data," because HIV infection was not reportable back then. "But we count AIDS cases." He said, "Fine. Tell me how many AIDS cases are women." It was like a 10:1 ratio at that time, and I had a 1:1 ratio of HIV in the clinic. So he said, "Well, then, Quinn's crazy. How can this be happening?" He wrote in his article that he was not sure where Quinn got his statistics, but they do not jive with the state health department's statistics on AIDS. Therefore, women do not need to worry about AIDS as a heterosexually transmitted disease, period.

Now, Jim Curran—well, not so much Jim, who was very supportive of my initial reports on this, because the CDC was not buying this in any big way either—no, it was Harold Jaffe who was my antagonist at first. He and I have become good friends since. But I asked Harold, why did he fight me so much on that. He said, "Tom, I will tell you one main reason. You said that, just as in Africa, where there is a heterosexual epidemic, HIV can spread among high-risk heterosexuals, be transmitted male to female, female to male. You used the phrase 'heterosexual population.'" He said, "The press does not hear the words 'high risk.' They hear the phrase 'heterosexual population,' which is 95 percent of the population. And you were saying just as in Africa, which has this huge epidemic, that this was going to be a huge epidemic here in the States." He said, "They're not listening to those few words." So he said, "I have to counter that and tell them, 'It is not going to be a big general heterosexual epidemic,'" which I never said it would be. He said, "They just do not hear the five words." He said, "So I know they won't hear that, so I won't even say it at all. I will just say, 'We are not seeing what we call tertiary transmission.'" I replied, "Who knows what tertiary transmission is. First, male to male, that would be primary. Secondary could be male to another male who is bisexual, who then gives it to a woman. No, sorry. Male to another male, who spreads it to a woman or something, who then spreads it back to a man. That would be tertiary. Or injecting-drug use being the secondary, which then eventually, without injecting-drug use, it is transmitted heterosexually. That would be tertiary." And he just said that, in terms of AIDS cases, we were not seeing heterosexual transmission yet. This was early. See, we are talking about 1987 or 1988, and not a lot of people really understood HIV serology. The only reason I knew it was because that is all I had been doing in Africa with Jonathan Mann and Skip Francis. All our studies were that way. And it finally caught on. Then everyone was doing serologic studies. But not in STD clinics. So that is why *The New England Journal of Medicine*, I think, took our report, because it was controversial, and there were letters to the editor and things like that. *The New England Journal of Medicine* likes that kind of stuff.

Harden: We will stop here today. But I want to say that your entire coverage of this is most interesting, because, of course, we are back, then, to the differences in mission of the NIH to do research and the CDC to do the epidemiology and to be the liaison to the public as to what is happening.

Quinn: Right.

Harden: And the words that we use. Tony Fauci got himself in lots of problems over his attempt to be very scientific...

Quinn: Yes.

Harden: ...and say that they were 99 percent sure that it was not casually transmitted, and then the newspaper said that Dr. Fauci says it can be casually transmitted, because there was a one percent chance that it could be…

Quinn: …transmitted, because a doctor will never say never.

Harden: That is right.

Quinn: That is the bottom line. We will not.

Harden: But I think this whole linguistic problem in terms of research and medicine and the public, and the translation of information from one to the other is an extremely important part, and this epidemic has just highlighted it.

Quinn: Yes. In my work, the divisions between the NIH, the CDC, and so forth, were gray divisions. We overlapped immensely. And I am epidemiologically trained from my time in Seattle. But I am also laboratory trained to do basic research from my time at the NIH. I try to put the two together. And the CDC has laboratory people there that do basic research. We all overlap one another. When that overlap is beneficial and we are complementary to a certain degree, working together to solve a problem, then that is fine. When it causes lots and lots of friction that is antagonistic and does not solve the problem, that is bad.

In the case of Projet SIDA, it was complementary. There was friction because of the two institutions. I think it is the history of the institutions. But it was complementary. We put people together there that wanted to come up with answers to questions, solve questions, I should say, and it paid off, I think, in the long run. It was, to me, a very rewarding and satisfying type of project, one that I, for the rest of my life, will be extremely proud of. When people bring up the friction, I say, "Yes. But when do not we have friction?" I have friction down in the laboratory next to me. The NIH has it from laboratory to laboratory. Life

is filled with frictions. But do not focus on the frictions. Focus on the products that came out, and whether they hold the test of time.

Of everything that Projet SIDA published, none of it has ever been retracted. It all holds true. The data are there. The specimens are there. The Africans worked with the Americans and with the Europeans. It was a truly international collaborative arrangement that worked. It shows that if you have a big enough problem and enough interested parties, they can work together. Yes, they will have their fights, but they will still work together. So when people say that the CDC is to do this and—yes, that is their goals and their mission. But the individuals within that can overlap one another and complement each other, and to me, that has been the exciting part.

Harden: Very good. Let us stop here for today.

***This is the second interview with Dr. Thomas Quinn. The date is 16 December 1996. The interview is being conducted in a conference room, Building 31, National Institutes of Health, Bethesda, Maryland. The interviewers, again, are Dr. Victoria A. Harden, Director, NIH Historical Office, and Dr. Caroline Hannaway, NIH Historical Consultant.***

Harden: In our first interview, Dr. Quinn, you were summarizing the activities of Projet SIDA and the African experience. We have a couple more follow-up questions on that before we move into a discussion of your later work.

You noted that there was a problem with the ELISA [enzyme-linked immunosorbent assay] assays in Africa because of people who had malaria, and that this caused problems with ascertaining the prevalence of AIDS. Could you give a fairly non-technical but specific explanation for what was going on and how these kinds of tests work? People do not perhaps understand why a test might not be specific.

Quinn: Early on, as the test was developed, it became clear that there were a number of biological entities that could give a reaction in the ELISA test but would not definitely reflect HIV infection. In the United States, it was learned that pregnancy, for example, would give you a false positive ELISA. Then you would do what is called a Western blot, and that would show whether viral antibody was present to viral proteins, so it would differentiate the false positives from the true positives. The ELISA was made to be exquisitely sensitive so that it would not miss any positive tests or positive infected people. But this was at the expense of its not being as specific; that is, it would give false-positive reactions so that it would not miss any true positives. So it became clear that you had to take a Western blot, an immunoblot, and run the same patient sera on the immunoblot. If there were specific antibodies to the

virus, they would react to the proteins, and that would then confirm that the initial ELISA test was positive and reflect a true infection.

On the other hand, if there was a false-positive reaction, then those viral proteins would not become visible on the immunoblot, and you would tell the person that they had a false-positive test and they were not truly infected. It pretty much became clear very early, within the first six months of licensure of the assay, that it was very sensitive but not that specific, and that you needed a two-tiered test to confirm the result.

When this test was brought over to Africa, the problem was compounded several-fold, because there were a lot of endemic diseases that caused an immunologic reactivity that would come up positive on the ELISA test. But then when you went to do the Western blot, it would be clear that these were false positives, not true positives.

Harden: May I ask just one quick question? When you did an ELISA test on someone, did you tell them that they had a positive result and that a second test had to be done?

Quinn: No.

Harden: Or did you just go ahead and do the second test?

Quinn: You had to go ahead and do the test. Especially in the early days when we were really just getting our feet wet with the ELISA. We wanted to be sure that when we told someone they were HIV-infected, that it was a confirmed positive. So everyone that we informed that they were infected went through the two-step process. Their sera was first tested by ELISA and then tested by the immunoblot, Western blot, and if both tests were positive, they were told they were positive for HIV.

There are a number of endemic diseases in Africa that will tip off the ELISA but not the immunoblot to be positive. We could go down a whole list of parasitic diseases, but malaria was the most notable one. There were even several papers written about that. There was also trepanosomiasis, there was filariasis, you name it, there was a long list. Even endemic bacterial infections, tuberculosis, things like that, could make the ELISA go positive. So you needed the Western blot. It became sort of a standard for scientific conduct there, that you had to do both tests. You could not get away with just doing the ELISA.

Later down the road, it became clear that this was too expensive for African countries. They could not afford to do both tests. For research purposes you

could, because that was funded just as it was in the United States, with the same kind of standards. But really to screen the blood supply in Africa, which was critically important since 10 percent of blood donors were positive, you had to have another way of ruling out those that were truly infected and those that were not. I became very closely interested in this whole question of what was the best sort of testing to do there. I will take you through a couple of steps.

The very first thing that we found out was that the blood in Zaire was being transfused within an hour of its being donated. You did not even have time to do the ELISA, never mind the Western blot. So what we did was work with some manufacturers to come up with rapid tests that could be done in 10 minutes and which could tell you whether the person was infected or not. It turned out that these tests were very, very good. In fact, they were actually a little better than the ELISA up front. But you still could not tell the person they were infected on the single rapid test—there were several rapid tests that we used—and there were at least three or four that we tried out.

Harden: What kinds of tests?

Quinn: The very first one that we used was called—I am blocking on its name now. It was made by Du Pont, and I keep wanting to say HIV. It was HIV something, HIVNET or something like that.

Harden: Was this the latex test?

Quinn: That was not yet. The latex test was actually the second one. Then the third one was called the Testpack, which was made by Abbott. The latex was made by Cambridge Bioscience, and the HIVNET—it is not HIVNET but it is similar to that; I am sorry I cannot think of the name; I could find out for you—and that was made by Du Pont.

What we did in a couple of different African settings, actually, was to utilize these rapid-screening tests and see whether they worked, and, in fact, they did. But what was a nice algorithm was that, since you did not have time to get the Western blot or even the ELISA done, you could use two separate rapid tests in tandem. If one was positive, then you confirmed this with the other one. That was equal to doing an ELISA and a Western blot, and yet you had your answer in 15 minutes and the blood could be transfused if the result was negative; if it was positive, the blood would be discarded.

The other part of trying to limit the amount of infected blood that was getting transfused to people was to teach the physicians that you should not transfuse individuals unless they desperately needed that transfusion because of the high

risk of getting infected with HIV. We set criteria that you should not transfuse the blood until a person's hematocrit or their anemia was at a certain level. That, in fact, cut the numbers of transfusions by 50 percent; just by doing that, we cut the need for the blood transfusions. Then we told them how to bank the blood. The Germans came in and wanted to work with us on this, and they built a brand new blood bank at Mama Yemo. We set up the rapid test, and so the blood could be banked while the testing was completed. In fact, during, I would say, 1988 to 1991, towards the latter years of Projet SIDA, we had a complete, effective blood-screening program in Kinshasa, so that infection was being transmitted in very few cases due to blood transfusion.

Hannaway: Was this organization of testing then moved to other African countries or elsewhere?

Quinn: It was, yes. Nairobi, of course, could afford it, and so could Zimbabwe and Harare, and a few other countries clearly had some financial support to help get these into place. The Swedes were interested in working with African countries. And so were the Germans and the British. It became an international effort. This was one thing the international community could do for Africa—to get down there and start screening the blood supply—because up to 10 to 15 percent of all HIV infections in Africa were due to blood transfusions. That became very clear early on. The rest was all heterosexual transmission, but 15 percent was through blood transfusions.

So the international community reacted to that either by building blood banks so that the blood could be banked while it was properly screened, or going in with these rapid tests. I became very interested in the rapid-test format, and our laboratory was the first one actually to bring the rapid test into blood-banking scenarios in Africa.

Later, we were to take the rapid tests into epidemiologic studies; that is, a lot of people that we enrolled into our prospective studies wanted to know the results of their test right away. They did not want to come back several weeks or months later. In some cases, we were afraid we would lose them. If they had a positive test or a negative test, we could counsel them immediately, before they left the clinic setting. So we went into this tandem screening process: screen with one rapid test, confirm with the other, give the patient the result.

We have subsequently, although we have not published this yet, done this in emergency rooms here in the United States, so that people who have a tendency to come in for medical care but may not come back for the results of tests can get an answer right away.

Then, of course, we would always, even with these two rapid tests, still go to a Western blot eventually. But we would tell the person preliminarily if they were positive for this infection.

Hannaway: So the technology transfer was back to the United States in this instance?

Quinn: Yes, it was. Actually, it was tested out more in Africa and then brought back to the U.S. to put to more practical purposes.

Hannaway: I think we have covered most of the situation in Africa, unless you have anything else that you wish to add about that.

Quinn: I am sure we could talk for hours about Africa. I think we should bring Africa up to the present date, because we have talked about the history: Where are we now? The situation has not really improved very much. Even after 15 years of working in Africa, we are faced with an epidemic that is still spreading, still increasing.

There are hopeful signs that condom distribution programs can work, that treatment of sexually transmitted diseases can work, and one of those studies—actually, both of those were done in Kinshasa, one in Tanzania, and we are doing one in Uganda now—shows that with an aggressive educational campaign linked with a treatment campaign of sexually transmitted diseases, one can limit the spread of HIV. But, unfortunately, it is only happening in very select areas that it is being piloted in. Whether the rest of sub-Saharan Africa will be able to afford these kind of campaigns is not clear, and it is probably not realistic to think so.

What is really needed for Africa is a vaccine. In my heart of hearts, I know the only way we are going to beat this in Africa is with a vaccine. We can do all these other things, they will have modest effects in slowing the epidemic. But when you already have 20 percent of your population infected, in places like Uganda, Rwanda, and Malawi, no matter how effectively you slow the transmission, it is still going to be there because there is just such a critical mass of people carrying the virus and who are infectious to their sex partners or through the blood donation system or through dirty needles. It will only be with a vaccine, I believe, that we will be able to stem the tide of this particular epidemic.

It does not mean we should not work on the other aspects. I think we need to keep the education level up and all the other interventions. But I think we are at a critical stage now in Africa where so many people have become infected that it will have a tremendous demographic impact, it will shorten the lifespans of

people, it will have an enormous economic impact. That is, the people who contribute to the economic health and well-being of a country are going to die before they can contribute to that well-being. You will end up with declining economies, which increases the poverty situation, which, again, is part of the vicious cycle that seems to spur on the spread of HIV, sexually transmitted diseases, and civil unrest, as we saw in Rwanda, and the cycle continues.

What is scary about Africa right now for me is that we are seeing this virus emerge in countries where it had been relatively quiescent. Nigeria is the most recent example of that. Nigeria is the most populous country in Africa. In the 1980s, there was nothing going on there. We looked. There was very little virus, and we never understood why, with so many people there. But it was very limited. In the 1990s, it is a very different story.

Hannaway: And South Africa?

Quinn: South Africa is the next scenario. Then you have tuberculosis as the second leading epidemic, linked onto HIV. Both are steadily increasing. The tuberculosis epidemic is very problematic because we are getting drug resistance, it is occurring in people who are HIV-infected, and it is probably the leading opportunistic infection in Africans who are HIV-infected. I think we are at a very dangerous, if I can use that word, point in time with the AIDS epidemic, because it has gotten up to such high levels in this population that unless we come up with something very dramatic—I do not mean a little something, I mean very dramatic, and that is the vaccine—the situation is only going to get worse. In terms of the world, on a per population basis, when you talk about the magnitude of the epidemic, Africa will always have the greatest burden in terms of prevalence of the population infected. We will talk about Asia in a minute.

Hannaway: Yes.

Quinn: Asia may have a lot more infected people in the next few years, if you weigh it, but there are 2 or 3 billion people in Asia, and there is only half a billion in Africa. Even though the numbers of people with HIV will be greater in Asia, per 100,000 population, it will still be worse in Africa, and I think people have to keep that in mind. And the countries do not have the resources to fight it without help.

Hannaway: Maybe we should now talk about AIDS in other developing countries.

Quinn: Right.

Hannaway: You started publishing on AIDS in India back in 1986?

Quinn: 1986 was my first trip to India. In the context of what we have been talking about, Haiti was 1983, I guess, and so was Africa. Both occurred in 1983. In 1984, we got Projet SIDA up and going. I started working in Nairobi along with the Canadians and other Americans there. We also started doing some work in the Caribbean as well—besides Haiti, also in Trinidad and Jamaica, with some other Americans in those settings. It was clear that these were the areas hard-hit with HIV.

But nothing was happening in Asia. It was very quiet. There was some talk about it going on in the Philippines, but it was the American servicemen in the military that were infected, and there was a concern it might spread. But it was very limited. There just was not much going on.

Then I was asked in 1986 to take a look at some blood samples from people to see if they were carrying HIV infection. I said, "Who are these people? Who is it coming from?" It was from some prostitutes in Bombay, in Madras. I said, "Sure, I would be glad to look at them." I was asked because the blood was coming to the NIH and I had an established laboratory that had done work in Africa and could deal with these "auto-antibodies" that might give you false-positive reactions. So people said, "Why don't we have Quinn's laboratory take a look at the samples, because if it is anything like Africa, he will be able to tease out who is truly positive and who is not."

Out of the several hundred samples that we tested, 10 were positive, and he was very disturbed by this fact. He said, "We do not have any AIDS cases in India. How can we have infection?" He said, "I want to see the blots." I remember this very distinctly. So I came down to NIH with the blots and I showed him. I said, "These are all HIV-1 infected. There is a concern about this new virus; HIV-2 could also be here, and we are going to work on that." It did later turn out that there was also some HIV-2 in India. But in these samples, they were all HIV-1 infected.

The samples had come to me from a leading scientist in Vellore, India, and that was Dr. Jacob John. He was the one who actually originally obtained the samples. So we communicated the results to Dr. John, and Dr. Ramalinga Swami went back to India and announced to Parliament that HIV infection had arrived. The reaction was, "It can't be. There are no AIDS cases here. How can we have HIV?" We would try to explain, "That is because after you first get infected, it takes 9 or 10 years, and then you develop AIDS." We already knew by 1986-87 that there was a prolonged period of infection. They just said, "It can't be. Your tests are wrong. False positives. We have heard about this

malaria cross-reaction. We do not believe it." We said, "Well, then, allow us to continue testing, and we will see if it is true or not."

By 1987, we had proposed a prospective study of female prostitutes in several cities in India, and this was done in collaboration with a colleague named [Dr. Richard] Dick Kaslow, who was here with NIH and just left a year ago. He is now at the University of Alabama. We put together the proposal and submitted it to both the United States and Indian government for funding. The United States government approved it, and would have given us funding. The Indian government said, "No way. We do not have AIDS, we do not have an HIV problem. Your tests are all wrong, and we are closing the door on this. Our policy will be, we will keep foreigners out of our country. By keeping foreigners out, we will prevent HIV from getting into this country." Period, end of story.

That was 1987, and nothing happened for probably two or three years, literally nothing, except HIV continued to spread, but unknowingly, to the Indians, or to anyone else, for that matter.

Meanwhile, in Bangkok and in Thailand, the American military, the Army, was doing some prospective studies, and they found the same thing that we found in India, which was, in 1987, a very low level of infection. But by 1990, it had already risen to 10 percent of sex workers and 10 percent of injecting-drug users. By 1992, it was up to 40 percent, so it was really escalating in Thailand.

There were some Indian scientists back in the late 1980s who were believers that these original tests were right but who were prevented from doing anything because of this government shutdown on HIV. But by 1991, they were becoming a little more forceful, saying, "We really need to do something." And I happened to go there in either 1991 or 1992 for a scientific meeting, and I was approached by the Indian government. It had changed over. There were new people in the ICMR. They said, "Remember that project you wanted to do back in 1987?" and I said, "Yes." They said, "We are ready. Can you put it into place?" We said, "Four years have gone by. But, yes." We answered yes, we would do it. Then the question was, where? Where should we set up a project with the Indians to study this epidemic?

I was in Bombay at the time, and I went into the hospitals to see what kind of patients they had, and it was like revisiting Haiti, back in 1983. It was the same scenario. Here were all these AIDS cases, all these Kaposi's sarcoma patients, all these people with herpes that was eating away at genitalia; tuberculosis was running rampant. They already had a TB problem, but now with HIV, it was much worse. The Indian government said, "No. Bombay would be one place,

but we want you to look at another place where we have a big commitment to virologic research, and that is in Pune.

Pune had an institute called the National Indian Virologic Institute, or NIV for short, so I traveled up there and visited with the director, who was very willing to set up a project. So we sat down to talk. Now, the timing of this was very interesting for me as an individual, because Projet SIDA was closing out as a long...

Harden: In ninety what?

Quinn: 1991. It was closing out, and here I was in India and the Indian government was saying, "We want you to set up another Projet SIDA, but here in India with us." Here I had committed my life's work to the Zairian project. Now that was nonexistent except for getting specimens out to continue the work. So I had time and energy, and I had some staff that were willing to work in India.

So I remember calling—this is a funny little story—but I called up one of my former fellows who was working at Hopkins. His name was Dr. [Robert] Bob Bollinger. I forgot the time difference [between Pune and Baltimore] and I reached him at 4:00 a.m. I said, "Bob, would you like to come out to India and do a project here?" He had previously worked in India. He knew a little Hindi. So he immediately said, "When can I get there?" and I said, "How about in a week. Pick up a portable computer on your way and we will write a grant while you are out here."

He arrived within two weeks, sat down with the director of this institute, punched out and submitted a grant, and it was due within about two months. It was ranked number one of all the grants submitted for doing international AIDS research. He was listed as principal investigator. I again played the role of facilitator and helped to establish the laboratory aspect of the project. It got started, I guess, in 1992 in Pune. And we have been working there ever since.

After the first year, a Dr. Tripathy, a very senior individual, became the director of the Indian Medical Council for Research, or the Indian Council for Medical Research, ICMR. He said, "Let's build an AIDS institute. This is enough. We are going to have a big, big problem." So he had the foresight to see that. In fact, we do not have an AIDS institute here. And he wanted it centralized. He wanted one AIDS institute to start out with, and then, depending on the epidemic, he would set up other ones in other countries.

Harden: But was this AIDS institute clinical, to take care of patients, to do research, or both?

Quinn: To do research. The problem with clinical care of AIDS patients in India was that they did not have the drugs or the money to pay for them. Just like in Africa, once you were diagnosed with HIV, there was not much to do about it.

What he wanted to know was this the same virus in India? Who was susceptible? How was it spreading? What can we do to stop its spread? And can we make a vaccine here? Because we were in the 1990s and everyone was talking vaccine, vaccine. That is exactly what he did. He set aside a new institute, built it from scratch, called NARI, National AIDS Research Institute. And it was the sister institute to NIV.

Now, the then-director of NIV was not very happy about this. He thought it should all be part of NIV. So there was a political struggle about this. In India, everything is very political. But the two institutes function separately. At the present time, each one has its own director. Hopkins and the NIH are affiliated with NARI at this point for AIDS research. Now, [Dr. Robert] Bob Purcell here at NIAID, who does hepatitis research and other virologic research, is still working with NIV. So NIAID itself works with both. I work for NARI and Bob Purcell and others with NIV. Then Hopkins got pulled in through this Dr. Bollinger to help facilitate the U.S. funding.

What the Indians said they would do is match the U.S. money. So if the United States gave $500,000 a year, which is what the funding was, the Indians would put in $500,000. But in fact they put in more because they built the big institute. It is a beautiful institute. It was completed in 1993 or 1994, so it has been occupied for the last two or three years. Their scientists go to Johns Hopkins under a Fogarty International Training Grant, and they shuttle back and forth. We basically take four of their scientists every year, and they either get an MPH [masters in public health] in epidemiology or they get laboratory science instruction in technology transfer. In fact, the director of NARI just came over and spent six months with me and Dr. Bollinger and Dr. Fauci in Dr. Fauci's laboratory, and he just returned. Dr. Gadkari is his name.

So there is a very nice working relationship between the Americans and the Indians on this project. Now we are not the only ones. Other projects have started to develop in Bombay, where the epidemic is very bad. There are a number of other groups working there. The University of Texas has a group working out there with a Dr. Hera. Other sites: Dr. Jacob John, still in Vellore, is building an AIDS program there. We have been into Hyderabad and also a couple of other cities where we have been looking at truck drivers. Again, truck drivers played a key role in the spread of HIV in Africa. We wanted to see if they were doing the same in India, and the answer is yes, they were. There are

very high rates of infection from truck drivers as they move from town to town. They are basically responsible for the movement of the virus into the rural areas of India. In the urban settings, where we have been working, it is all heterosexual transmission. In the north, in a state called Manipur, it is injecting-drug use. Manipur borders onto Burma and surrounding areas as part of the golden triangle for heroin exportation, and so HIV is very widespread in that area.

Although it took the Indians maybe four or five years to recognize that HIV was there and spreading, as the government turned over with different scientists, different appointees from the ministries and so forth, a recognition came about that there was a problem and they needed to do something about it.

The sad part is that back in 1986, when we first started, there were only a few infected people in India, a country of almost a billion people. It was almost a negligible epidemic. If they had alerted the population, tried to implement some very vigorous educational campaigns, they might have been able to slow it. But by waiting four years, the epidemic had already gone from just a few cases to probably two million, that fast, because of the sheer population. Now it is at 4½ million in India. No other country in the world has as many HIV-infected people at this time as India does, and it has only been there for the last eight or nine years. So it is a very sad situation in which the Indians could have learned from others.

But India should not be singled out. Almost every country that I have worked with from the very beginnings of its epidemic, if the numbers of AIDS cases are low, does not think they need to put many resources into fighting this epidemic. America is no different. We started out with AIDS cases because we did not know HIV was about. But after you learn that HIV is the cause, then you start doing surveillance for HIV, and you go to the government and say, "You have got infected people. Do something. Start a campaign." They say, "But we only have a few cases of AIDS. We are not as bad as you in America." That is the normal reaction. We were just in Malaysia. They gave us that same reaction; Indonesia, same reaction. And many of the African countries during those early years of the epidemic were saying, "You are only here to point the blame at us, but your country has the most cases. You should have the blame." It became a finger-pointing contest in many of the early years of this epidemic. I think that has changed. We are not doing that as much.

Harden: Do you think that is primarily because of political expediency, the people in power at a given time do not want to deal with it? Or do you think it is lack of understanding of the science and the medicine of how it works? Is there a way to change this?

Quinn: It is definitely the latter. In India, it was a definite misunderstanding about this disease, that HIV could take that long to become positive. They also really thought that Asians were resistant to this epidemic. I remember in 1986, they said, "Look, we are the only area in the world that does not have any AIDS cases except those that got infected in the United States and came here to die." It was true, if you looked at all of Asia, in two to three billion people, no one had AIDS in 1986, 1987, or 1988. It was almost nonexistent. Back then everyone was saying, "I will bet you there is a genetic resistance and that the Asians have that genetic resistance." That is not the case anymore. So there was a misunderstanding of the science. Then the second was, it was political expediency to deny that there was this big problem because you would have to put resources into it, and you had limited resources.

Hannaway: Resources are finite.

Quinn: Right. Also, it went against the grain of the country to say, hey, we have a heterosexual epidemic of HIV. No one wanted to admit that. Certainly, I know from visiting in Malaysia, which is a Muslim country, by and large, that prostitution is not supposed to occur in a Muslim country. Injecting-drug use? That cannot occur. Well, it does whether people like it or not. There is travel abroad, there is poverty. Poverty and travel interlinking brings HIV to the poor, where they are trying to service people's sexual needs, if that is what you want to call it, and certainly Thailand knows that. They have learned their lesson, and they have closed many of the brothels down as a result. Injecting-drug users are getting certain types of rehabilitation to try and slow down the spread of HIV through injecting-drug use.

There are many lessons that one learns about society from this epidemic. I think it reflects many of the faults of the worldwide society, and it also reflects the cultures of individual countries, because AIDS spreads differently in different countries, depending on what their culture is. For us, in the 1970s, we had this free love, come-out-of-the-closet, gay-life time, and that is what caused AIDS to spread like wildfire. As we moved into the 1980s, we had a real drug problem. It was not as big in the 1970s. But in the 1980s, we had a real drug problem. You know that from the number of visits to emergency rooms due to crack cocaine. There were approximately 10-fold increases everywhere in the mid-1980s. As that becomes part of your culture—and that is one of the ways HIV gets spread—it moves into that. It changes the face of the epidemic. When you go to Thailand, you have two things happening in their culture. One is a drug trade, which is up in the north of Thailand along the Burmese and the Chinese border. It is the golden triangle and so forth. And you have a sex trade. So, as the virus moves in, it quickly gets into those two high-risk groups and that

becomes a part of the epidemic, first drugs and then the heterosexuals. Why it took so long to get there, I do not know, because that culture has been like that for the last 20 to 30 years, or even longer.

Hannaway: The sex trade really developed in conjunction with the Vietnam war. Thailand was a place for soldiers from Australia and America to have R and R [rest and recreation].

Quinn: Right. I do not understand why it did not take off sooner. It is well known that Europeans, Americans, and others go to Thailand and engage in some of that sex tourism, I guess it is called. But HIV was not a problem until the late 1980s. It really took a much longer period of time to start. That was not true for the Caribbean, South America, the Americas in general, Europe, or other places.

We have been talking about what is happening in Asia, and it is very interesting working in Japan as well, because the Japanese right off the bat said they did have an AIDS problem, but it was all in their hemophiliacs and it was due to exported blood from the United States. They were absolutely correct. The beginning of their epidemic was solely in hemophiliacs, and it was due to blood products from the U.S. prior to our implementation of screening. Tens of thousands of hemophiliacs got infected. For the next 10 years, that was all they said.

But meanwhile, as HIV was starting to spread in Thailand and neighboring countries, it was also starting to get into Japan. For the early years of the 1990s, the Japanese refused to acknowledge that any Japanese nationals were getting HIV-infected because they just did not think their nationals would engage in this sex tourism or anything. They said, "HIV is now in two groups in our country: foreigners and hemophiliacs, and that is it." It has only been within the last year that Japan, a very well-developed, economically well-off country, has come to the realization, "Oops, it is now in our own nationals due to heterosexual spread, drug use, and back and forth from other infected people, the hemophiliacs to their spouses." They clearly now recognize it is endemic in their country. But it took them so long to realize that, because it went against the grain of their culture. Their culture does not want to admit that there might be promiscuity or a drug problem.

Harden: How does that compare to the Communist Chinese? Do you have data on that situation?

Quinn: The Chinese are very interesting. Now I have not been to China, but a couple of my colleagues have. What is interesting in China is that China has readily admitted they have a problem with HIV in Hunan Province, which is a southern

province. It is part of the golden triangle. It is the southern border of China. They are worried about the area neighboring Hong Kong. It is called the Guangxi Province. I think I am saying it right, Guangxi. But the Chinese now say that they probably have anywhere between 50,000 to 100,000 infected people, and two years ago they said they had none. So they have come to the realization that it is moving in. It is moving into hot areas, those being the injecting-drug users through the Hunan Province and then some of the sex activities—I do not want to call it sex trade—but there is that element to it near Hong Kong. As China takes over Hong Kong, that could be a concern.

Also, Vietnam has recently admitted they have a problem with HIV, and it is from drug abuse and prostitution, remnants of the Vietnam War or whatever. It is interesting. This is not to say that as they open up borders and they start to become part of the global society, that HIV comes with it. But HIV does get there through international travel. That is how it is moved around different areas of the world. As you become a more open society, you put yourself at risk for further spread of HIV.

The one country that remains relatively closed has been Cuba. Cuba does have HIV, but it is much more limited in its dimensions than anywhere else in the world. But it is a very restrictive type of society in that if you are identified as HIV positive, you are put off onto a farm.

Hannaway: The Cubans have taken restrictive measures to try to contain AIDS.

Quinn: Right. Now, I did not get into that, but 1986 was a very popular year for me to travel around, and so I went to Cuba on the behest of PAHO, the Pan American Health Organization.

Hannaway: Yes.

Quinn: The Ministry of Health had put in a request that an AIDS expert with international experience please come down and look at their program, and I was asked to go. Actually, I think Dr. [Anthony] Fauci was asked to go, but he was too high a level official. He was a director. He was an assistant surgeon general, and that was too high a level to go to Cuba back in the 1980s. So Tony asked me to go. I went and spent a week there, and I reviewed their program. I met with Fidel Castro and their Minister of Health, and spent a fair amount of time going over their program. In fact, I would not support it. I got yelled at quite a bit by Fidel at that time.

Harden: Tell us more about the situation in the Cuban AIDS camps, the quarantine camps. I would like to hear you compare the situation of caring for patients

versus limiting their freedoms in Cuba with the situation in other countries, both in the Caribbean and the United States.

Quinn: First, in our society, restriction would not work in any event because it is against our constitutional rights for freedom to be restricted in a situation where a person's individuality is sacrificed for the sake of the society. You might say that could be justified if it was just one or two cases. But once we knew we had an AIDS epidemic, we were well into the thousands to close to hundreds of thousands of cases, and it was too late at that point even to take those kinds of restrictive measures.

Now those measures have been taken in the past with tuberculosis and other transmissible diseases in the hope that their spread could be limited from an infectious point of view, like, say, if it was an aerosolized pathogen or something. With HIV, it is a sexually transmitted or a blood-transmitted pathogen, and to try and restrict a person's sexuality or something along those lines is obviously extremely difficult. It goes against our culture. So when we look at the Cuban situation, we would say it is wrong, period; that you cannot take away a person's rights to live in society.

Harden: Some people have said that is what should have been done.

Quinn: Early on, you mean. If it was only a few cases, maybe you could have pulled that off, if you could have struck a balance with the person's individual rights and so forth. It is which weighs more, the rights of society versus the rights of the individual. In our society, we like to see a nice balance. But, preferentially, we value the rights of the individual. We do not say that those should outweigh those of society, but that they should be balanced.

In Cuba, it is the rights of society. The individual does not count, period. That is their culture. And while we can view their culture as being different than ours, they have adopted that culture. Now maybe some do not want it to be that way any longer, but they did in the 1970s and 1980s. That was the kind of culture they wanted. It was a culture in which society dominated over the rights of the individual. So from their perspective, it made all the sense in the world. They only had a few hundred cases at most. Those you could remove from society and place in a quarantine situation where they could still live up to society's standards, but they would be removed from the risk of transmitting HIV to other people outside of that environment. So for Cubans, it fits within their societal culture. We can criticize that because it is different than the way we want to live. But if it is the way they want to live, then it is their right to make that judgment.

Harden: Now, here is a complicating situation. The Guantanamo Haitian detention center was run by Americans, but has been criticized very strongly in terms of how the HIV-infected Haitians that were detained there were treated. The U.S. military has come under criticism for forcibly injecting Depo Provera, I think it was, to induce abortions if HIV-positive women were pregnant.

Hannaway: That was the assertion.

Harden: Yes, it is an assertion. These are things that give a civil libertarian pause. I wondered if you knew anything about that situation.

Quinn: I do not know a thing about that. All I can tell you is what took place in Cuba during those times and that it is still ongoing. There has been zero word out of there as to whether it has been effective or not effective.

There were many logistical problems with their approach in that they were going to screen the entire population of, what, 10 million Cubans, and that would take three years. They thought that by the time they got to the end, they would have removed everyone who was infected. But the problem was that the ones that you screened in the first year that were negative now could have gotten infected, and so you then have to repeat the process. It is a continual process of testing everyone every few years. It is not an inexpensive undertaking.

The other thing that was interesting was the source of most of their HIV. Everyone asked, "Where did it come from?" A lot of it came from sort of their nationalists. I forget; there is a name for them. But they would go out and preach the Cuban socialistic system to places in Africa and other parts of Central America and South America. Those internationalists, I guess they were called, when they came back, sometimes they would be infected. Obviously, again, human nature being human nature, and with their being away from family for prolonged periods of time, they would engage in sexual activity with someone overseas and get infected.

Hannaway: This is a strong subtext to this whole discussion, isn't it.

Quinn: What is?

Hannaway: The sexuality that is unacknowledged by almost every society, and yet that is clearly how the disease spreads.

Quinn: It is part of our human nature. It is part of our biology as human beings that we are sexually active.

Hannaway: But it is inconvenient for the political structure.

Quinn: The political structure has a very difficult time dealing with it. Depending on the morals of that particular society, they have an extremely difficult time reckoning with this particular problem. That is what I was saying about what took place in Malaysia per se. It is a Muslim country. You are not supposed to be promiscuous in that society. You certainly are not supposed to offer sex for pay. But that is what happens. It is there. I am sure that these types of attitudes occur in areas of the Middle East and elsewhere where sex for pay is even more verboten and yet it probably occurs—maybe not as commonly as in other places, but it does occur.

Hannaway: Iran would be the most interesting case.

Quinn: Yes, that would be. But very little is known about that region of the world.

Harden: To return to Cuba, would you comment on the assertion that it was primarily Cuban soldiers in Angola who brought AIDS back to Cuba.

Quinn: They had brought some HIV-2 back, actually. One of the first HIV-2 cases that was seen in the Western hemisphere outside of the United States was in Cuba, and it was a soldier who had visited Angola. But, no. It was more than that. They have what are termed internationalists. They are not soldiers. They are well-educated people who go to other countries and try to help. Other countries will say, "How do you have such a good health-care system in Cuba?" It is one of the best in the world. To Castro's credit, he really did set up a very good health-care system. People would say, "We want to set something up," and he would send out these internationalists.

Hannaway: So these are the political troops.

Quinn: That is correct. I do not know how much of that occurs now, because their economy is so stricken. I have not heard much about what is going on in Cuba for a long time, other than the little politics you hear about from time to time.

Hannaway: Sure. Well, what about Brazil?

Quinn: Brazil?

Hannaway: Yes, please.

Quinn: Brazil is interesting in terms of its epidemic. It mirrors the United States quite a bit. Their first cases were recognized about two to three years after ours were,

and they were solely among gay men. It was clearly a homosexually transmitted disease there. Then, over time, we started to see more heterosexual transmission. It was really in the 1990s that we started to see it. So it is similar to our epidemic that way, in that we are starting to see more heterosexual spread.

But what is interesting about some of the Latin American countries, Brazil in particular, is that you did not see spread from injecting-drug use. In the United States, you went from homosexual to injecting-drug use and then to heterosexual spread. It was three successive waves, and some of those waves are still reaching their peak. But in Brazil, it went from homosexual to heterosexual spread. It did not tie in as much with injecting-drug use as it has in the United States.

Hannaway: Why was that?

Quinn: I cannot answer the question why do they not have as much drug abuse as we do. It may be money, it may be the variety of drugs available. They may be more into crack cocaine kind of things, but not injecting heroin like the East Coast of the United States is into. But more important is the Latin component that homosexuality was never fully acknowledged as a sexual mode of life. It was still the macho Latino way of life to be a heterosexual, but people having homosexual tendencies would often do both, and so you had more bisexual men. There is a much higher proportion of bisexual men in Latin America than there are in the United States, probably because of this cultural constraint that they could not be solely homosexual.

Hannaway: Homosexuals would be married?

Quinn: They would be married, right. So you had a lot of bisexual relations going on, and that is how HIV moved into the women pretty quickly. Now their epidemic has taken on quite a heterosexual component too. But I do not think it is yet as bad as that of the United States. As much as most people do not even want to recognize that we have a heterosexual epidemic here, I think it is worse here than it is in Brazil per se. But Brazil would probably be number two in the Americas for the numbers of cases reported in women. They are quite close to the United States in terms of their financial ability to care for some of their HIV-infected people, and so it is not quite to the same level. But I know a lot of the drugs, protease inhibitors, AZT, and other drugs, are there and are readily available to HIV-infected people.

We are still studying how HIV spreads among gay men there. I am part of a group that is working in Rio de Janeiro—that is the only city I have actually

been to in Brazil—where we have set up this project. It is only a few years old, so I do not have a lot of answers on what is happening. But HIV is still spreading among gay men there. It has not slowed down too dramatically. It is still on the rise.

Harden: Can you say whether the situation in other South American countries is similar?

Quinn: I would say that it is quite similar. Although there are exceptions. In the Andean countries—Argentina, for example, and Peru—there is a lot of injecting-drug use. I do not understand the cultures well enough as to why some have a lot of injecting-drug use and others do not, but Argentina has a problem with it. As a result, something like 60 to 70 percent of all their AIDS cases are among injecting-drug users, so it is a big problem for them.

Even in our own country, I do not understand why the East Coast is predominantly injecting-drug use and heterosexual transmission, and the West Coast is predominantly, if you look at the distribution of risk behaviors, still a lot of gay-transmitted disease, homosexual spread—some heterosexual, some injecting-drug use, but not nearly the escalation in these types of spread that we have here in this side of the country.

So, as you go down the Americas, you find different countries having different problems in terms of which risk group is spreading the virus at a greater rate than others. Mexico I know very little about in terms of their kind of distribution, although I have some information on that that is coming in from PAHO.

But the Americas as a whole are all very similar in having the same kind of epidemic that we have. It is just a proportional kind of difference, slightly more injecting-drug users than heterosexuals. I tend to lump a lot of the Latin American countries into the same phenomenon that we are seeing. In fact, we have a lot of Latinos in our own country that reflect what is going on in those other countries. So I do not really see a big difference there.

Where I really see the major threat of this, the future of the epidemic in terms of continued fast escalation, is Africa and Asia. The Americas will still be bad, some countries worse than others, with Haiti certainly being one of the worst. But Haiti is so much like Africa, in many ways. Its politics, the civil unrest, the rates of infection, the poverty, things like that, are very similar to what you see in any African country that is been hard hit by this epidemic.

Harden: Before we turn to more global and speculative kinds of questions, is there any of your other current work that we should discuss that you can think of?

Quinn: No. I think we have covered most of it.

Harden: This project has focused on the intramural program, and yet we have found, especially in your case, that what is happening in the intramural program at the NIH has implications around the world. Could you comment, using AIDS as an example, on the NIH as a place to do research, on the intramural program and what it offers, and perhaps what its limitations are?

Quinn: That is a tough question. What the intramural research program enabled me to do, on very short notice, was quickly jump into areas in which HIV was rapidly spreading at the earliest level point. Case in point: Haiti. If I had not been in the intramural research program, I am not sure I would have been able to have been part of that endeavor. So it was due to the connections of being here and working with people that were high up in our government that, when the Haitian government made that overture to let us work together, quickly went to Dr. Krause, from Dr. Krause to me, because I had had some of this experience already of working with AIDS patients and having expertise in sexually transmitted diseases among gay men. I was at the right place at the right time with the right training. Being in the right place was being here intramurally. If I had been somewhere else, I am not sure whether I would have met Dr. Krause and that I would have been tapped to go and do that.

I can show that several-fold, the instances of where I was able to get moving. For Zaire would be the next step. Having met Peter Piot previously did help, obviously, in terms of getting the invitation. But being able to get the funding within just a few months through Dr. Krause and Dr. Sell enabled me to set up that project at very short notice. Once we realized what was going on, it enabled me then to set up a long-term project.

If there is a difference between intramural and extramural research, it is that if something is happening hot and fast and needs immediate investigation, an intervention type of scenario, that can be done quicker in an intramural environment where the funds are available. It may need shifting some of those funds around, but the funds are there. In an extramural environment, you need to write a grant. You need to wait nine months. You need to get it reviewed. It needs to have pilot data if the peer reviewers are going to accept it, and that takes time. If I had been in the extramural environment at that time, I would not have had enough time or pilot data to convince anyone to get going. So it is expediency that I think the intramural program facilitates. The same is true at the CDC, they can respond to an emergency outbreak, wherever in the world it is, because that is what they are targeted to do. And the money is set aside there or it is shifted around according to priority. So, obviously, the intramural

program sets the priorities for the research that goes on. It is reviewed by scientific counsel and so forth. This was felt to be a blossoming epidemic that needed not just epidemiology work, but biomedical basic research had to beset into place as well.

The other advantage of the intramural program is the collaborations that it allows. The NIH is often the place on the cutting edge of science. That was true in this case with Bob Gallo and people such as [Dr. Malcolm] Mal Martin working with specimens and trying to figure out what the cause of AIDS was. In my case, I interfaced with Mal Martin, who got me in touch with Montagnier, and so it went that route. But it could have been an interface with Bob Gallo as well; it could have gone either way.

I would say that, when you look at it retrospectively, being in the intramural program enabled me to respond to a research need in a very rapid fashion. It was able to provide the funds or resources that were available to address a series of questions, and it worked for me. It also enabled me to have connections with the people who were being asked to move quickly into this area—Dr. Krause for one, Dr. Fauci for the other. Those are my two main contacts, and also my two main supporters for international AIDS research.

Harden: One very brief follow-up question. What kind of an overall population of AIDS investigators are we talking about? You mentioned how you had the right training and were in the right place at the right time. How many other people would have had all of those qualifications and be ready to step in? Do we have hundreds of people like this? Do we have three? Just a general sense.

Quinn: In the intramural research program?

Harden: In the United States.

Quinn: I would say it is limited.

Harden: That is what I would say.

Quinn: The intramural research program is very basic biology research, with some applied clinical research, but it is limited in that latter aspect. When you consider the third sort of research, which is biomedical epidemiologic research, there is an even smaller group. When this AIDS epidemic came around, the numbers of those with expertise in working with gay men would have to be essentially two or three.

Harden: We are down to a couple of people?

Quinn: Yes. It was very small. On the extramural side, there are more people, obviously, because that is how I was trained in the extramural environment as well. I have training in both, but that area was not available in the intramural program, so I went extramural. They picked it up there and then came back and brought that expertise to the intramural program. So, in that case, extramural held down intramural. You could say maybe it worked the other way as well because, as I have stayed in the intramural program but been based in an extramural environment, I have been able to make NIH intramural contacts out to the extramural area. So it has been entirely a two-way street for me. It has been the best of both worlds.

Hannaway: And that is an unusual situation?

Quinn: It is very unusual. The NIH does not have many other examples of this type of setting. It is true, though, that two of their institutes are based in Baltimore, adjacent to Johns Hopkins. In fact, the [National] Cancer Institute used to be affiliated with the University of Maryland. And I would say our institute, NIAID, for example, has reached out and has set up relationships with George Washington University and with Howard University, where there is some cross-fertilization in terms of expertise for clinical experience, for basic biology research. I think the same is happening between Hopkins and the NIH. Certainly, I think Dr. [Harold] Varmus has approved this new genetic center to be based in Baltimore in affiliation with Johns Hopkins. It is a joint undertaking where scientists, both intramural and extramural, are going to be working side by side. I do not know a whole lot about this, but my understanding is that more of this sort of gray zone of where intramural ends and extramural begins will start to overlap to some degree. I think that is good. It has to be done carefully. There cannot be favoritism. But it is geographic location, the NIH is based in Bethesda and Hopkins is nearby, and so are Howard [University], George Washington [University], Georgetown [University], and so forth. There are a lot of close ties. When you have the University of Washington being 3,000 miles away, it is hard to get those same kind of relationships.

Hannaway: You had the component in developing your own setup that a bigger clinical population was needed.

Quinn: It was.

Hannaway: For research.

Quinn: Right.

Hannaway: Than was available in Bethesda.

Harden: I have been writing down some speculative questions that I would like you to address if you would. I had a conversation at a meeting with someone who was analyzing military needs into the next century. He was talking about shifting political power in the Third World, especially in Asia. I raised my hand and said, "I know there is a tremendously high infection rate of AIDS among many soldiers, especially in Southeast Asian armies. What is that going to do to their military and the way they are thinking?" He said, "We haven't thought about that." He just did not know how to answer. It was an issue that he had never addressed. Would you care to address what the population impact and the political impact around the world will be from this disease?

Quinn: It is hard to see into the crystal ball, obviously, as to how things are going to be 10 or 20 years hence. Within the foreseeable future, as we mentioned, AIDS will be having a demographic impact in Africa, and it probably will in Asia, but not many people have died from the disease in Asia yet. There are infected people, but they are still serving in the military for two years. Usually in the military there, you serve from the ages of 18 to 20 or 22 or 24, and then you leave. Even if you became infected at age 17 or 18, you are still healthy and you can fight for your army and do those things, so that, militarily, the immediate impact is not that dramatic.

The biggest worry that we have always had was about the loss of intellectuals from smaller societies due to this disease and who would fill the gap. In other words, as HIV affected some of the brightest and energetic members of a population and they are killed off, who is left to fill that gap? That leads to some political instability in a sense. So there was always a worry about that. And that worry dates back—I know this individual you are talking about did not think about it, but I can tell you that the CIA and other members of the military have been thinking about it. I have been interviewed by them, so I know that. Certainly back as early as 1986, I remember an interview with a couple of people from the CIA who were concerned about what was happening in different parts of Africa. The State Department has been very concerned about it.

But in Africa and other places, will there be depopulation? The answer is no, because there is a high fertility rate, and the population growth rate was already quite high. So, yes, population growth rate might decrease, but it will not go below zero, which is what needs to happen if there will be depopulation as a result of this epidemic.

Overall, the demographic impact will be there, but it is not going to be that

overwhelming, I do not believe. We will probably, if other countries follow what has happened in the U.S., get to a certain point where the infection rate starts to level off. It does not necessarily decline, but it starts to level off. There is a saturation level, and each society and each culture will determine its own saturation point. I think we are starting to reach it now in the U.S. Every year, we lose 70,000 people due to AIDS, but we gain 70,000 new infections. That is stability. That does not mean the epidemic is going away. It just means it has reached a level of stability. That is where we are right now. The number of new infections equals the number of deaths, so we are in a state of equilibrium.

Harden: In the United States?

Quinn: In the United States. Will that happen in other countries? I think it probably will. You get to a point where a certain number of people are dying and a certain number of people are getting infected, and then the prevalence rate does not go up any higher and it starts to level off. But that does not mean that the number of new infections is going down. It just means that they have reached a certain level that it cannot saturate much more than that, because it takes 10 years to die from the disease anyway. Maybe that time period will get longer in some countries, but in Africa and others, if anything, it is shorter than 10 years. It is certainly not longer.

I do not believe that we are going to see half of the military decimated by HIV. I think that in other countries like Thailand, they will screen. The positives might be excluded, but there will be many uninfected people. They might have to recruit more people into the military than they usually do to keep their numbers up. But I actually do not see a military destabilization due to HIV in the next few years. I cannot say what will happen in 10 years. That is just too far down the road.

Hannaway: The destabilization that you have referred to in Africa is more due to the death of people in the prime of their lives?

Quinn: Right.

Hannaway: And the loss of their economic potential?

Quinn: I think it is the economic, and to some degree the intellectual, base that could be severely hampered by this, because as the economy weakens, there is less money to educate people.

I just met with the Rwandan ambassador to the United States on Friday, and we talked for an hour. He said, "Our country has now been stable for two years.

Our refugees are returning." And he said, "We have nothing. We have zero left." Now, 20 percent of that population may also be HIV-infected. He does not even know. He has no health structure left. He has nothing. And he is here, going around from agency to agency, university to university, saying, "What can you do to help us rebuild our infrastructure?" In a lot of ways I worry about what HIV will do in situations like that.

We worried a lot about that in Zaire. We saw some of the bright people infected. Because people with money were often in the intellectual group. They were leaders of business and so on. Because they had money, they had more wives. The more wives they had, the more likely they could have gotten infected, and then they would die from AIDS. I worry more about that and about their declining economy being much more of an instability factor than I do about the military having HIV-infected people.

Harden: But you lose the transmission of culture and skills if the people die, so that your younger generation may not...

Quinn: Right, learn.

Harden: And then be able to step into the shoes of their elders nearly so well. So that is a problem. Another thing, as I said before, that has been a subtext of all this is the fact that AIDS is a sexually transmitted disease. Now, we have dealt with syphilis and gonorrhea and other sexually transmitted diseases in the past.

Quinn: Not very well.

Harden: Not very well. And we know that no matter how many times people have been told to abstain from sex to keep down the transmission of disease, people do not do so.

Quinn: No.

Harden: I wondered in this case, given how deadly this disease has been, whether we are either going to see people's behavior change radically on the one hand or whether the religious, social, and political structures on the other hand accommodate to the reality any better?

Quinn: A little of both, but not completely. First, there was radical change in the sexual behavior of gay men, period. End of epidemic in gay men. The disease became endemic, but was no longer epidemic. It can happen if people are motivated enough.

Harden: And they understand.

Quinn: And they understand it. The gays had their own community, they were self-motivated, well educated, leaders in our society, they had a voice in our government, and they said, "We have to do something. We are losing our populace." Their populace, their community, was getting decimated. It did not mean that the whole of American society was being decimated, but their community was. Within two years, their incidence rate dropped like a lead balloon, I guess you would say. So that is an example. It can work.

Thailand. Thailand is a combination of both, the best example of both that I can think of. One, men started to use condoms more. They did not want to get infected. The education message got out there. We have men that will put on two or three condoms so they do not get infected. But the government helped in this scenario. The government did change. They said, "All right, we have got to stop these sex trades," and they closed all the brothels. Yes, those involved in the sex trade moved to other places. These people did not go away overnight. This is their living. It is part of their culture. It is how some of these women build their dowry, or they go back to their families with the money they made as young teenage girls. But, by and large, the government put its foot down and said, "What are we going to do?" They are building an aggressive STD intervention, and treatment program; and they have their education, condom-distribution program. Both sides worked. Sexual habits changed. People started using condoms more. There was a decline in the frequency in which the men in Thailand visited prostitutes. And the government cut down on the legalized part of prostitution.

Will that happen in other areas? Probably not. It is not happening in our own country, and I am not sure it will happen in most others. Something happened in Thailand that really helped motivate that country to get moving, and something happened in the gay community in the United States really to get that going.

Harden: And Australia has had a very good record, has it not?

Quinn: Now, AIDS in Australia has had a different aspect to it. They believed in the needle-exchange program, and that was adopted into law there. They were able to cut HIV transmission among injecting-drug users in half within six months by enacting that kind of rule. Their epidemic was primarily homosexual spread as well. Because the infection rate went down in the gays like it did in the U.S., and it went down in injecting-drug users, they do not have a third epidemic yet. They do not have a heterosexual epidemic because it has slowed down in the other two segments. So they have been very effective in that.

Other European countries have had mixed successes, just as we have had some mixed successes. But those are probably the most outstanding success rates.

Now, with regard to Africa, we talked a little about the need for a vaccine, and we do need one and the situation is desperate. But, in the meantime, what are they doing and are there success stories? I alluded to that. Yes, there are. In Tanzania, there was a very small pilot program where they did syndromic treatment of STDs; that is, if you had a symptomatic discharge or pain on urination or an ulcer, you automatically were treated, right on the spot. Doctors were educated to go out and to do this. In other villages, it was left as the status quo, which was hardly anything. They showed that the villages with the intensive STD campaign had a 44 percent reduction in the spread of HIV compared to the other control group. Then, in Uganda, they are doing a mass treatment campaign of STDs to see if the rates of both can be lowered, and I am involved in that particular study. We will have to wait to see what the answer to that is.

But in the meantime, while we are awaiting a vaccine, there are only three basic tenets that I would put out, and the first is education. Let everyone be aware of the problem, know how to protect themselves, and be very universal in informing everyone. The whole population needs to hear the information. Get the government involved, get your community leaders involved, get your NGOs [non-governmental organizations] involved. But that is the educational campaign.

The second is that it should be linked with a very aggressive STD treatment campaign. Get STDs treated, you will lower the transmission rate because you will lower the efficiency of transmission, and you can link that with counseling about HIV and STDs and how to protect yourself.

Then the third would be a condom distribution program or microbicides for women; condoms for men, microbicides for women. The studies on microbicides are not all in yet, but I link them with condoms. When I say condoms, I want it to be known that I am also talking about female-motivated ways of protecting themselves, and that is the microbicides right now. The female condom has not panned out. It is not popular.

Harden: Not popular?

Quinn: No. It is there, but it is…

Harden: Not being used as they had hoped?

Quinn: Right.

Harden: I have one more speculative question to ask you. In the fourteenth century, the Black Death decimated the population of Europe and changed many aspects of society. In your opinion, is AIDS a natural phenomenon that will bring the human population into a more sustainable size, given the earth's resources? If we figure out a way to vaccinate against AIDS, will something else take its place? Is there any link, as some people have suggested, between the elimination of smallpox and the advent of AIDS? Do you see what I am getting at with these kinds of questions?

Quinn: Yes. It is sort of the chapter I am supposed to be writing for Dr. Krause and Dr. Fauci right now, but...

Hannaway: It is the political economy of disease.

Quinn: Yes, exactly. I will give you my perspective, but I am still thinking about the question and my view is still evolving.

One, AIDS is an emerging disease. It is still emerging. It is emerging in many different societies and different cultures and countries. Why did it emerge? Where did it come from? I think it was changes in our population, in our society, that brought it about. Yes, we have had prostitutes and sexuality going on for eons. Homosexuality has been around forever and so forth. But why did this hit in the 1950s, or 1960s, whenever it started to emerge? I think it was that we became much more of a mobile society and, particularly in Africa, there was a lot of movement of populations from the rural to the urban areas. There was a lot of change in the economy. They really just hit their industrial revolution. It is barely beginning there. If you think about the industrial revolution in Europe you see the emergence of tuberculosis, syphilis, and other diseases that occurred as people moved into the cities. I think that is what happened in Africa and, because it happened at a time that we hit the jet age, the 1950s and the 1960s, people were flying everywhere and meeting different people, different societies, and the interchange of sexuality, almost, and the spread of a new sexually transmitted disease. How it got from monkey to man or whatever may come down to an accidental slice of monkey meat, since they eat monkeys quite readily in Africa, blood getting on someone's hand and then getting infected into that person, and then spreading it as that person went to market and in the evening met six women, and who knows what happened. I think we will probably never know for sure the answer as to its origins, but it is clear what got it to move around the world so quickly, and that is our mobile society. So our society is responsible for it.

Will something take its place? Will we eradicate it? Right now, because of AIDS, we are seeing the emergence of other diseases or re-emergence of old diseases. You only have to look at the list of opportunistic infections, with tuberculosis heading the list. It was coming under control; now it is back up again in incidence, and it is re-emerging. We are also seeing the spread of other diseases, cryptosporidiosis, microsporidiosis, and other types of opportunistic infections. I would say that as our society continues to evolve and our populations change and our weather changes, our temperatures of certain things...

It is similar to the question of where did legionella come from? Legionella has been around forever, but then we built air conditioners and it liked air conditioners. Then it got into the ventilation and spread.

What about Ebola? Ebola, fortunately, is a very short-term disease, so it does not have as much time to be mobile. AIDS did, and I think that was the unique aspect of AIDS. In 10 years of carrying a virus, you are going to move around, you may have sex with more than two people during that time, and so you spread the disease. With Ebola, you are dead in 14 days. It is kind of hard to spread it. And you are sick most of the time, very sick. Whether that will emerge, or diseases like that, we will have to wait and see. We worry about dengue and the hemorrhagic aspects of the disease, and that has spread as we have moved the vector, the mosquito, around.

I think as we tamper more with environments, we tend to change a little the ecological niche of microorganisms. As they get disturbed or we come into close contact with them, we will see new diseases. It is like cyclospora and strawberries, that was a classic example. Or cholera getting into the jet planes' water systems. Did you read about that one? That was amazing. I think we are going to continue to see that.

We are better at recognizing it now, too. This was probably going on years ago, but we missed it. People got sick, got better. That was that. Now, if you get 10 people with diarrhea, it is an outbreak. Let us go investigate it. And you find out that it is a new disease or an old disease that has come back.

Harden: Will AIDS reduce the world population to the same extent that the Black Death did?

Quinn: No. It does not even come close. No. Flat answer.

Harden: Do you have figures predicted as to how much it might reduce the world's

population?

Quinn: I think the growth rate of our population will compensate for the increased deaths.

Harden: So you think not. Okay.

Quinn: Yes, basically. I think, in the next 10 years, we may lose 50 million people from AIDS. How many billions of people are we? We are talking global. Ninety percent of this disease is in developing countries. I do not know our world population number. What is it at, three or four billion? Some say six.

Hannaway: I thought it was over three.

Quinn: Yes. It is over three. I know that. Well, you take 50 million—is that what I said? I hope I said 50 million deaths in the next 10 years. I do not want to say that, but I think the reality is that is probably what the numbers will be by 2006. But the growth rate will compensate for it, so that the absolute population, the number of people, will still be greater in 2006 than it will be in 1997. There will be an increase. Will it be the same increase that would have occurred if AIDS had not been here? No. It probably would be 50 million greater. But 50 million out of 5 billion, or whatever the number is, if you do the math. It is not even a one percent decline. So, no, I do not see that.

Now, the population was very small when the plague broke out, and it did decimate the population of Europe.

Harden: Twenty-five percent is the general estimate.

Quinn: Right. That is not going to happen here. I just do not believe it will happen. Even in Asia, where we will have 10 million people in the next few years infected with this virus, 10 million out of probably 2 billion in Asia is a very small number. You have to keep that in mind. It is still a lot of deaths. And as an infectious disease, it is going to become the number one killer. I do believe that. Well, tuberculosis will probably still be called number one, but a lot of those cases are going to be due to HIV. But when you put it in a perspective of the whole society and other diseases and the overall population growth rate, I do not think the impact of AIDS will be listed by historians, 10 or 50 years from now, as equivalent to the Black Plague of the European Middle Ages. When was it exactly?

Harden: 1349.

Quinn: Yes. It just is not going to be as historical as that, except that we learned a lot about the spread of disease from AIDS and we have learned a lot about society and culture. If it does bring about a cultural revolution, which is what [Dr.] Jonathan Mann and others would like to see happen as a result of this, then it will be listed as a historical event.

Harden: The Black Death is often looked at as marking the end of the Middle Ages and the beginning of the Renaissance.

Quinn: Yes, that actually is.

Harden: And historians were all asked for analogies when AIDS first came: Is this the new Black Death? Of course, no one knew what was going to happen in 1983-84. But it was a speculative question that journalists asked us. You are convinced that the absolute numbers could not come anywhere near the present situation.

Quinn: They are not going to come anywhere near this. No. I do not think they will. The same thing, and everyone always tried to draw an analogy to the influenza pandemic in the beginning of this century.

Hannaway: 1918.

Quinn: Right. Nothing compares to that in terms of the rapidity with which it killed people. I was not alive then, but from what I understand, just about every household lost one or two people due to the flu. It was in New York and other...

Hannaway: People often died within a week.

Quinn: Yes. Whereas this is going to hang around for ages, and I do not know if it is going to go away very soon. The other thing historically I think we will learn a lot about from it, where this retrovirus will make its mark from a biological perspective. We have never had anything quite of this nature before, that could establish an infection, not kill the host, hang around for 21 years, then kill the host. It is not just hanging around. It is eating away at the person. But the fact is that it takes 10 years. I think that realization will open up avenues into other chronic debilitating diseases—multiple sclerosis, the Jacob-Creutzfeldt diseases, all of these. Probably Alzheimer's disease may turn out to be a virologic disease that takes 60 years. Who knows? I think it has opened enormous insights into our ability to interact with microorganisms.

Harden: So, you do not see anything mystical about the fact that the first human retrovirus was identified in 1979 and AIDS appeared in 1981.

Quinn: No.

Harden: We could see, we recognized it when it happened.

Quinn: Exactly. And that gave us the ability to recognize it. But did that have anything to do with its origins? No, because I know it was going on earlier than 1979 in Africa. We have a virus from Zaire in 1974.

Harden: That is interesting.

Quinn: 1976. Sorry. Take that back. Several viruses. They are the first isolates. They were not recovered in 1976. They were recovered in 1986, actually. But a team of our investigators, Projet SIDA, went up to a very remote area of Zaire, where Ebola is endemic. They went back to this village and found, in fact, that there were people there whose blood had been saved and which was HIV positive. They recovered the virus directly from them, from the saved blood samples. Some of those people had died in the 10-year interval, and a couple were still alive. They show that they still have that same virus. So HIV was here before we knew about retroviruses. I know it was around before 1976. There is just no doubt in my mind it is been there for at least an additional decade. How far back it goes in man, I do not know. I stop in the 1960s. Maybe 1959, there is one report.

Hannaway: In Manchester, England?

Quinn: Yes. Oh, no, it is not Manchester. Actually it is a blood sample that was saved from someone in Africa, and people say that it was antibody positive. I do not know if that is true or not. Yes. Then there is this man from Manchester. I do not know anything about whether that has been validated or not. I just know there is a lot of debate over it.

Hannaway: I have a couple of questions that come back to you personally, I think, to wind up here.

Quinn: Okay.

Hannaway: Obviously, AIDS has had a profound effect upon your career.

Quinn: Yes.

Hannaway: In numerous ways...

Quinn: Emphatically yes.

Hannaway: ...which you have detailed.

Quinn: Yes.

Hannaway: Would you just comment on: How do you think your career would have developed if AIDS had not come along?

Quinn: Everything I was doing before AIDS brings me up to 1981, which is when the AIDS outbreak began, and I started to shift a little responsibility over to that. I probably would not be doing the degree of international work that I am doing, even though that was my love at the time and I had been trained and really wanted to do that.

Hannaway: Your work in malaria and...

Quinn: Right. But I had shifted away from malaria after I had worked out in Seattle. I was doing sexually transmitted diseases. When I got to Baltimore, before AIDS, I was setting up research programs on chlamydia and other sexually transmitted diseases, gonorrhea and so forth, herpes. I had a herpes project. I would say my career would have gone the route of research in other sexually transmitted diseases. Maybe that would have taken me to other countries or not. But it is because of AIDS that now STDs have gained international notoriety. Before, there was just an old dermatologic disease. Let us not talk about it. Those are dirty things and no one is supposed to talk about them. Now it is quite politically correct to talk about them. So that probably would have helped facilitate my work in STDs as well. But without HIV, that is where I think I probably would have been now. In terms of intramural versus extramural, I cannot answer that. I certainly enjoyed working intramurally before the AIDS outbreak, and I think we just would have seen how things had developed. There are people at the NIH who work in STDs other than HIV. [Dr.] Stephen Straus never got into HIV in any big way and is a major-time herpes researcher. He joined the same time I joined, and we probably would have linked up more and done more intramurally on herpes, gonorrhea, chlamydia, and things like that. I might have been working on a chlamydia vaccine five years ago instead of next year or whenever it comes around.

Harden: What about your family and friends? We have talked with a number of people and some people have reported that their children, especially teenage children, did not want to talk to friends at school about the fact that their parent worked with HIV. Others did not have any problem. What was your personal experience?

Quinn: The complete opposite. My kids begged me to come to school and talk to their friends about AIDS. And I do that. Annually, I go into at least three or four different elementary and high schools and spend half a day at each of those schools teaching each of the classes about AIDS, what we know about it, how we can protect ourselves, and what is new in the science of AIDS. They love it. My son did a science project on the multiplicity of HIV. He was learning logarithms and things, and so he showed how the HIV virus can replicate in a logarithmic manner, and he pulled my papers and other papers from my files and so on and worked on that. That was at the age of 10.

So, no. My children got indoctrinated to AIDS as a biological infectious disease just like any other virus or bacterium is. You get vaccinated against some of them. We do not have a vaccine for this, so we had better know other things on how to protect ourselves. Very early, they just accepted it as part of life, that it is there and that it is a horrible disease, but there are ways to protect yourself. So they have been very proactive. They have been very proud actually to have me come into the classroom and to teach.

Harden: You have never had any of your colleagues, as some people have, not want to shake your hands in the early 1980s or any experiences like that?

Quinn: No. People would say to my wife, "How do you deal with him traveling to all these different countries and the diseases he might bring back?" It was more that than it was, "Aren't you worried about getting AIDS from him because he works with AIDS?" That aspect rarely came up, unless it was a very paranoid person who was very suspicious about how this was spread.

But, right off the bat, by the time I was off to Haiti, we knew how this thing was spread. We knew it was not a respiratory pathogen that I was going to inhale, and we knew that I was not going to get it by drinking the water or things like that. You knew you had to have sex to do it, and even then, no one believed it was heterosexual, so no one was even worried about that aspect of it. Unless you were gay, a shooter, or a hemophiliac, there was not any other way you were going to get it. This was in the early 1980s. Obviously, as heterosexual spread came on, that was always there.

Then when the blood test came out, I made it clear to my wife and family that, because I did work with the virus and my laboratory worked with the virus, that we had a policy that our blood was tested every six months to every year to make sure we remained negative and did not convert over otherwise, or be a laboratory accident. But, yes, people were worried about me handling the blood and maybe cutting myself. They would say to my wife, "Aren't you worried

about that?

Hannaway: What did she say?

Quinn: She said, "I guess, but," she said, "it is not something I am going to dwell on. If it happens to him, it happens to him, but let's hope he's careful with it." Then she knew I was moving more out of the laboratory and had other people doing a lot of the laboratory work. That was by 1985-1986.

I am rarely in the laboratory handling the bloods now. I do draw blood from some of these people, and you can always stick yourself that way. But the odds of getting infected from an accidental needlestick, as you know, are three out of a thousand, and now, with AZT, that is even lower. So it is a low risk. Let us put it that way. I think my wife accepted that I might have a risk in working in the field that I work in anyway.

Harden: You were in infectious diseases.

Quinn: I was in infectious diseases. So I think she always knew that. She has medical training herself. She was in dental school and she took the same kind of classes I took on microbiology and so on. She is well educated. I think the more educated you are about this, the less paranoid you are about it. You know better what the risks and the issues are and can handle it mentally better than doing it from your gut, which is, "Do not come near me."

Harden: That is right. Is there anything else that you wanted to ask?

Hannaway: Just one brief question, to wind up. People have criticized the NIH for not responding quickly enough to AIDS. Now, in recent years, obviously, it has responded overwhelmingly. And you mentioned that you, yourself, had some problems in 1988 with the media.

Quinn: Yes.

Hannaway: Would you just like to finish up by commenting on whether you think that the NIH responded as best as it could given its institutional structure and organization? How would you characterize the response that is happening now? How do you try to inform the people in the press about the NIH's mission?

Quinn: I think it did respond appropriately, and I think it was fairly fast. For me, it could not have been any faster. I could not do any more. I went in, nearly 80 percent of my time was AIDS, and the 20 percent left was my little chlamydia program. Actually, I have cut back a little on AIDS to get back to my original

chlamydia program, because I am in a situation where I have lots of other people that are doing the work and I am more advising, thinking of projects, things like that. But back then, I actually had to do it. I did not have anyone to say, "Can you do this?" I had to do it. So I would say, the NIH did move rapidly.

Could we have had more resources and moved quicker? Yes. Did the government want to give us those resources and have us move quicker? No. Look at the CDC. They had a very tiny program set up, because we did not know it was going to be that bad. Then, when it was all in gay men, the politics back then was why do we need to spend all this money on some disease that is affecting a very tiny proportion of our society? That money is better spent on cancer, which is affecting everyone. That, I think, was the general reasoning. So, with limited resources, the NIH and the CDC did the best they could in learning how this disease spread.

I remember, though, all those comments, and I always thought they were unfair, because the people that were working full time on this did not need to have their heads yelled at that they were not doing enough. They were devoting their life to it. I must say, Tony Fauci had death threats against him half the time, and I think it was so inappropriate. Here was a man setting aside all his time to do it. Yes, he may benefit from it in terms of the science knowledge and the benefits he would get from that. But he decided to stake it out, it could have just fizzled, and then where was he at that point? But he did not. He said, "I am going to go after this." Cliff Lane: "I am going to go after it." Dick Krause, too. To some degree, myself, Mal Martin. People all along the gamut really went after it.

Could other people have joined us? Yes. It would have been nice. It would have helped a little bit. And people did show an interest. But no one knew what was causing it, so they did not know where to begin. Just to throw a whole lot of money at it without any idea of how to proceed I think would have been wasteful. So the funding for it gradually grew, and the number of people working on it. As more knowledge came about, more people could then make hypotheses based on that knowledge to test by the scientific method. I think that is the best way of going about this type of research.

That is eventually what has now panned out in the 1990s. We learned about reverse transcriptase, and now we have a drug that inhibits it. We learned about the protease inhibitor, and now we have a drug, a designer drug. They basically designed a drug to fit inside an enzyme and stop it. That is unbelievable. I think that has revolutionized medicine in many ways. It is like the astronauts, an astronaut campaign. Sure we went to the moon and back and that was exciting, but all the benefits of that program into our society are million-fold, and we do not realize it. You hear that an awful lot of the time. I think the

same is going to be true in medicine because of AIDS and HIV. So a lot will have been learned.

But just to have fools rush in and spend lots of money and come out with an answer on how to beat this was not going to work. It was just not. We did not have the technology. If this disease happened 20 years from now, we might have been able to say, "Hey, we know about retroviruses. We know about their enzymes. We can do this, this, and this." And within three years, we might have been where we are now. The problem was just what you stated before. In 1979, you discovered the first retrovirus; in 1981, you got an AIDS epidemic with a retrovirus. But all you knew was that it was caused by a retrovirus and nothing else about the biology of a retrovirus. So it just took time.

I think those discoveries will continue to accelerate. At least I am hopeful that they will. But biotechnology has to make those leaps, and it is not always going to be around HIV. It could be about something else, but then there will be an immediate crossover to HIV. And the reverse is true. We may make a major accomplishment in HIV and move it over to some other field and make an accomplishment there.

Harden: Is there anything else that you would like to say?

Quinn: No. I think I have talked for about five or six hours and I have probably exhausted my comments.

Harden: It has been a tremendously interesting interview, and we thank you very much.

Quinn: Thank you for having the interest in this topic.

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