This is the first oral history interview with Dr. Robert Gallo of the National Cancer Institute concerning the history of AIDS at the National Institutes of Health. The date is 25 August 1994. The interviewers are Dr. Victoria A. Harden, Director of the NIH Historical Office, and Dennis Rodrigues, program analyst, NIH Historical Office. The interview takes place in Dr. Gallo's laboratory in Building 37, Room 6A11, National Institutes of Health, Bethesda, Maryland.

Harden: Dr. Gallo, we would like to start by discussing certain aspects of the National Institutes of Health (NIH) when you first arrived in 1965. You have detailed your early education and your family life in your book *Virus Hunting*. Could you describe the size of the NIH campus, the environment at NIH for an intramural investigator, and perhaps some of the people who seemed to be leading figures at that time?

Gallo: I could describe the environment at that time, first physically, and then intellectually, and also some of the scientists who were here.

Physically I remember the campus as a rather beautiful place, with more trees than there are now. If I remember correctly, there was even a little brook that ran through part of the campus. There were many fewer buildings at that time. I actually lived next door to the campus. I used to crawl under a fence to get to work every day. Rather than going out on to Old Georgetown Road and coming in the normal route, I would just go into my backyard, under a fence, cross, and walk along a nice path among trees and with a little brook nearby to Building 10, the Clinical Center. I do not want to say the campus was pastoral, but it was suburban and still had a wooded area.

Geographically it was a nice place to work, and it was so convenient for me to live next door. Nonetheless, I had a feeling of the immensity of NIH, that it was something beyond me, that it was incredible that I was there. There were all kinds of activities going on that I felt that I would never understand, and all kinds of major scientific figures who were remote from me. So I was excited by the very fact that I was at NIH. But I did not have the feeling that I could ever really grasp, or understand it; as I said, it seemed immense to me, even though there were many fewer buildings.

The scientists that I had contact with, since I was in the Clinical Center, were, of course, principally clinical investigators, though Building 10 had then—and still does have—a number of basic scientists working in it. Clearly, in the second half of the sixties, the most exciting and visible research in basic science at NIH was [Dr.] Marshall Nirenberg's work. That was the time of the breaking of the genetic code. I cannot remember if that was the first project that I thought about, but Nirenberg was the first person I wanted to work with after finishing my Clinical Associate Program.

That was also the first time leukemia was cured. And I was on those wards at the Clinical Center. I had come from the University of Chicago to NIH not believing that approaches by combination chemotherapy could do very much for people with leukemia. I thought that it was not a very sophisticated therapy. It was not the kind of science that I wanted to be involved in. Then, while I was on those wards, with my own eyes I saw children beginning to be cured of leukemia for the first time.

NCI [National Cancer Institute] was not alone in this breakthrough. Other institutions like Sloan-Kettering [Cancer Center in New York] were heavily involved. But much of the pioneering work was done at NIH, certainly not by me, but by people around me. There was the development of supportive care for children who went into heart failure when they were given blood. The children were anemic. They had no platelets and they needed blood because they would hemorrhage, sometimes to death. The simple act of concentrating platelets, red cells, and white blood cells to fight infection allowed more children to survive and more intensive chemotherapy. It sounds simple, but it had not been done before.

People like [Dr. Emil J.] Freireich, [Dr. Ronald] Ron Janke, [Dr]. Emil Frei—and the people who worked with them—did that. It is almost a blur in my mind now; people were coming and going all the time. Those clinical investigators in NCI and leukemia research were major figures. Frei and Freireich left shortly after I came, but that work was continued by [Dr.] Paul Carbone, now Head of the Cancer Center at Wisconsin [University of Wisconsin, Madison].

[Dr. Vincent] Vince DeVita was away at Yale [University] for a sabbatical when I arrived. He came back to NIH a year or two later and started combination chemotherapy for patients with Hodgkin's disease for the first time. Concomitantly, [Dr.] Henry Kaplan was getting the first cures at Stanford [University] with radiation. Then DeVita's protocol, as well as those of some other people, began to get cures of Hodgkin's disease in that period of time.

Other basic scientists at NIH who were very visible that I was not connected with, but whom I certainly knew about, were people like [Drs. Christian] Chris Anfinsen, Sidney Udenfriend, and [Herbert] Weissbach. Then there were people in metabolic diseases whom I came to know well through the years, such as [Dr. Thomas] Tom Waldmann and [Dr. Donald] Don Chudee, in NCI's Metabolism Branch. I was rather interested in metabolic disorders so I followed some of their work quite closely. Two others were was [Dr. Robert W.] Berliner and [Dr.] Vaughn Weedon. Even though I was not connected to them—there was also [Dr. Donald] Don Fredrickson—I followed that work with quite a bit of interest. Naturally cardiovascular physiology—led by [Dr. Eugene] Braunwald—was universally recognized as an exciting laboratory at that time. From

the top of my head, those would be the names that I recall as most obvious to me then.

Harden: In your book you stated that the late 1960s was a time when many young M.D.'s in the United States idealized academic research. Then you said that this was a state of mind that seems to have gone the way of the dinosaurs. Could you expand on that statement?

Gallo: Yes. I do not see as much in young people anymore the attitude that I saw in myself and other young people then. Maybe it is a process of getting older and I do not see it because I am no longer a young person. But there seems to me to have been a decided change.

I was mentioning names of leading figures at NIH to you. I forgot the people in virology. I was not in virology when I first came to NIH. But a few years later there was [Dr. Robert] Bob Huebner, and [Dr. Wallace P.] Rowe. There was also the man who got sick with a neurological disease from a virus. I cannot remember his name at the moment. These were major figures.

I think that, for a person coming out of medical school, internship, and residency, it was very exciting just to see people with such reputations, and working in a place such as NIH was almost awesome for me. I do not know how to describe it, other than sounding naive, but I am not exaggerating.

Harden: This brings up the question of why did you stay at NIH and not go to an academic center?

Gallo: It is a little complicated because I always thought I wanted to go back to a university and do what I was supposed to do, which was the famous triad of teaching, research, and clinical work. I never thought that I would just do pure laboratory work. If so, what did I get an M.D. for? Was I not at a disadvantage having done so? Would I ever do what I could do best, after giving all that up? I never thought that I would stay at NIH and just do laboratory research.

What happened is that one year led to another and I became more and more removed from medicine. I began saying to myself, "This is what I really want to do." But I was still planning to go back to a university because I wanted to teach. If I was not going to be a clinician, at least I was going to be a teacher. But then the research work gets interesting, or exciting, however you want to describe it, and you become addicted to it and reasonably productive. NIH was a wonderful place to work. There was constant stimulation from so many good people, easy access to technology from so much diverse science around me, and the steadiness of funding.

If there are different kinds of scientists, then maybe I see myself as a gambler. There are many places where you cannot be a gambler because there is pressure for you to obtain year-to-year funding for your spending. But if you know you have money for five to ten years, or you are fairly certain of it, then you can ask longer-range questions in your scientific research. I thought that security of funding was the best reason for staying at NIH. I also believed then that at NIH you could bring ideas from the laboratory to the clinic as well as at any place in America, although I did not know that for sure. I do not know if that is true now. In fact, I doubt it. But, at that time, that was my thinking, and being in the Clinical Center added to it because I had very frequent contact with clinical investigators and clinicians.

But today, when I see young people, I do not get the feeling that they are so excitedly grateful that they are able to be involved in research and medicine. It is a given for them. I just do not see the same love.

Harden: You began by working with Dr. Seymour Perry...

Gallo: I am sorry. There is one other aspect of the attitude to research that I should comment on. I think that part of it also is the maturity of modern biomedical science. It is a business now. Once that happens, the age of innocence is over. People often ask—and have asked me, and I know that I ask many scientists—whether getting into industry, the developments in biotechnology, and the companies that have resulted, is good or bad? The answer is, "It is both." It is good because it catalyzes science, making it move forward to applications of research, which is the ultimate goal. I believe the good far outweighs the bad.

But the other side of the issue is that many scientists now have opportunities to be involved with companies. Young people will also see immediate opportunities in industry. In the period 1962 to 1965—when I was in training, or coming to NIH, there were great industrial scientists, but they were unusual. Most of the time, if you were an M.D. who wanted to do research, and you went into a company, you felt that you had failed in some way. There were exceptions without doubt, and it is important to emphasize this—many exceptions—but, in general, that was the attitude. Whereas today it seems as though going into the biotechnology industry is often a priority. That is a major switch.

Sorry, I interrupted your question.

Harden: That is all right. I just wanted to return to when you first arrived at NIH and were working with Dr. Seymour Perry. Would you comment on him as a mentor?

Gallo: Yes. Certainly, when you asked me about important figures at NIH, I should immediately have mentioned my own mentor. The reason that I

did not is that during my first year I was at a distance from him. He was in more of an administrative rather than a clinical position, so I did not have daily, or even weekly, contact with him during the first year that I was here.

It was in the later part of my second year that I had a lot of contact with Sy Perry, because at that time I joined a newly formed department which was called the Human Tumor Cell Biology Branch. This laboratory—of which I am now the Chief—the Laboratory of Tumor Cell Biology, is an offshoot of that, but it is devoted more to basic science. The time I spent with Sy Perry was my introduction to research on white blood cells.

When I was a medical student, I had worked with red blood cells, erythropoietin, and so on, but my introduction to white blood cell research, to cell kinetics, was at NIH. I think I could reasonably say that it was my first serious taste of biochemistry, or of getting into basic science. Sy had a very strong appreciation of the application of basic science to the study of the biology of the white blood cells. He had already formed, before I came, a connection with the Laboratory of Physiology. In the Laboratory of Physiology, there was—as there still is today—[Dr. Edward] Ted Breitman, who was closely connected to Sy Perry at the time as a Ph.D. biochemist. In a way I bridged the two laboratories as Buzz Cooper had done years before me. He is now at the University of Pennsylvania, or maybe he has moved from there. But, anyway, I just continued to do that kind of work.

This was also training for me in enzymology, as well as my first experience with culturing, obtaining, and handling human white blood cells and control cells from normal donors, PHA-stimulated lymphocytes. I remember those days very vividly. It was a time when people had just become able to separate lymphocytes from the neutrophils and the rest of the white blood cells. The method was by packing long columns with nylon fibers. The granulocytes would be trapped, and the lymphocytes would fall through. We began using normal lymphocytes stimulated with phytohemagglutinin, which had just been discovered by accident by [Drs.] Peter Nowell and Hungerford, I believe—or was it Nowell alone—at the University of Pennsylvania. They found that a certain plant extract, a certain plant agglutinin, could make human lymphocytes divide as a mitogenic factor mimicking—but being even better than—an antigen that a T cell was primed to. We barely knew the difference at that time between T and B cells. What I mean is that I do not know if we did know this at the beginning. I cannot remember whether we fully understood this.

I was comparing biochemically those PHA-stimulated lymphocytes to leukemic lymphoblasts from childhood acute lymphoblastic leukemia because this was a proliferating cell, a blast. We did a lot of comparative biochemistry. But it was a period when we did not have adequate notions of what to compare with what.

Today there are too many ideas. There are 100 things you want to compare. But back then you were just poking around saying, "Well, I think DNA is important. Maybe I should look at DNA polymerase as the enzymes that make DNA. I would like to study transfer RNA, because I believe that in translational control of protein synthesis—protein synthesis is important to the differentiation of a blood cell and leukemic cells do not differentiate properly—so maybe I should learn about transfer RNA."

Hence, my interest in Marshall Nirenberg was more than the average. I almost joined his laboratory, but then [Dr.] Sidney Pestka left Nirenberg's laboratory, came to NCI, and was working near me. I simply joined up with Sidney Pestka, and got a lot of training in transfer RNA molecular biology. It was my first taste of molecular biology, which was in its infancy at the time.

Sy Perry was a warm person who gave me opportunity, much liberty, and guidance any time I needed it. I would say he was as perfect a mentor as anyone could ask for, and far more patient than I, in a similar role, would ever have been with somebody like me. Sy always says that he would get to NIH early so he could avoid people and get his work done before the day started. He said—I did not remember this, but he said it at a party recently—that I would be at his door either before, or just after, he got in there, bothering him, asking for more of this, and more of that, more funding for whatever, and more space, and so on.

Harden: In your book *Virus Hunting*, you said that you learned, first about molecular biology, and then about skepticism in science, from Sidney Pestka. Would you expand on your comments about him and explain what you meant by that statement?

Gallo: Yes. I learned a number of things from Sidney Pestka. I have commented on Sy Perry and Ted Breitman, my first training in enzymology, and in basic science, more or less. What I did in erythropoietin as a medical student could be called basic science, but my first real biochemistry was with Breitman, and with new kinds of tools that I was not used to.

With Sidney [Pestka], I was extremely "professionalistic" and probably a little compulsive, too. What I remember best is that he never believed anything. A situation occurred which I described in the book and which I can never forget, because I wondered if I would be killed in the process. Sidney did not believe that the isotopes that were sent to us were necessarily what they were supposed to be. If New England Nuclear said, "This is labeled 'tritiated leucine,'" Sidney said, "Go prove it." We always had to do high-voltage electrophoresis on the isotopes. I had never done high-voltage electrophoresis before and I think that I am lucky that I did not end up staying in that group permanently as a mummy after doing high-voltage electrophoresis.

But one day I came to Sidney with some water. I said, "Sidney, how do we know this is water?" Because he was always saying we had to re-identify everything that we received. But, if you start thinking that way, you could never do any science, because you have to believe something at the beginning.

But I think Sidney's approach resulted from his experience in Marshall Nirenberg's laboratory where it was very important that they remove any traces of contaminating amino acids. They always subjected the substances that they were sent by some of the companies to such analyses.

Rodrigues: One question that always comes to mind when I talk to people who have worked in a laboratory and who, at a later point in their career, move into an administrative, or a coordinating role, is how do they feel about the transition? What did you feel when you moved out of the laboratory and were no longer working at the bench?

Gallo: The transition was gradual. Let me phrase my thoughts a little better. In moving from working full-time in the laboratory to not working there day-to-day, I cannot say that there was a specific moment when there was an emotion about it, because the transition occurred in multiple steps. It was not an all-or-nothing phenomenon. It occurred little by little until I finally had technicians or postdoctoral fellows joking with me, "Please don't use the equipment because you are not going to do it right." Then I started realizing that I was not spending my time best by being in the laboratory; it was better that I spend it talking to the postdoctoral fellows because they could do things better than I could. Of course, in time in a laboratory young people come in with new techniques that you have never done. When this occurs, the word that describes my feeling about this is insecurity.

It is not a good feeling when a young postdoctoral fellow has been well trained in a good laboratory in a technique that you have never seen done and that you need to know about. Then he or she becomes your mentor in learning such a technique, so that is not easy. This happens increasingly when technology changes as fast as it does in modern biology. There are always new techniques and there is always a young person bringing something new to the laboratory, so you have to become accustomed to it. After a while this insecurity passes and you try to give back what you are receiving. There are so many new things to learn from incoming people.

I would say that I did not have any traumatic emotions about going outside the laboratory and taking on a director's role. I do not like to call it managing. I hope to call it leadership. I think there is a difference. I have read the book by Stephen Covey [*The Seven Habits of Highly Effective People*] that is a best-seller. It said managers push people up the ladder, but a leader has to know that the ladder is on the right wall. I think that a

laboratory chief at NIH, if he is not working all the time in the laboratory, should be making sure that the ladder is leaning against the right wall.

I have to say that I was never overwhelmed with the need to pipette. I think there are people who need it for the serenity of it all. I am not one who feels that this is necessarily a great pleasure. I like seeing the results when they first come out when they do that.

Rodrigues: In 1972, when you were made Chief of the Human Tumor Cell Biology Branch, you renamed it the Laboratory of Tumor Cell Biology. This, in NIH's designation, indicated that there was a shift from a clinical to a basic science focus. Would you elaborate on your decision to emphasize basic science, and yet to emphasize studies that were closely related to disease problems?

Gallo: Yes. I changed the name of the unit. Of course, we were the administrative unit on it. The change from Human Tumor Cell Biology Branch to the Laboratory of Tumor Cell Biology was appropriate, because the laboratory was moving more towards basic science and away from day-to-day clinical involvement.

I felt—and I guess this happened to many people—that science was becoming more and more complicated. There was more and more technology, and it was becoming increasingly difficult to be good at this, and to be good at that, as well. I did not believe that I had to be good at everything. I just could not keep up with it. I did not think that I could be a good clinician and continue having patients and all the responsibility of that in Building 10, and also be able to do productive research that I felt was useful and exciting, and important and fun. You try to put all those things together and do it well, and, in addition, say, "I am also going to be on the wards." But, at a certain point, I did not feel that I could handle it all, so I had to make a choice.

On the other hand, I try not to forget my origins and the advantages that I have. I think the advantages M.D.'s have over Ph.D.s, if I may say this, is that they have a broader knowledge of biology related to medicine. I try to continue that. I thought that the NIH was the best place in the world to be at that time, because there was no doubt in my mind that it was unique in that scientists were able to do research that could be applied to clinical medicine.

I always thought that a good function of a laboratory at NIH—it does not have to be—was to have a theme, whether it was protein chemistry like Anfinsen, or the genetic code like Nirenberg. It is not exactly like a university department, but a branch or a laboratory at NIH is a sort of department with some independent investigators. The difference is that NIH laboratories have a theme, whereas a university department that has to teach all kinds of things does not necessarily have one.

I believed that the study of the biology of blood cells and their normal growth and differentiation would have ramifications for the understanding of the abnormal, for leukemias and for lymphomas, or for lack of production of blood cells—aplasias—or for deficiencies, like the immunodeficiencies that we find, like AIDS ultimately.

To this day, in my laboratory we study AIDS and we study leukemia and lymphomas. Sometimes we have branched out a little from those themes. We now do some research in breast cancer and obviously in Kaposi's sarcoma, but basically AIDS and leukemias and lyphomas still receive the emphasis in this laboratory. I believed that we could make our greatest impact by doing basic science, making our results known, publishing and presenting them, and hoping that they will get picked up for use in the clinic.

Sometimes this works well, and sometimes it does not. Let me give you an example. In 1993, the General Motors Award went to individuals from France, I think, and China for the retinoic acid treatment for acute promyelocytic leukemia. But I bet that very few people know where the first observation on this was made. It was made in this laboratory a decade earlier. So the discovery did not get picked up very fast. We established a cell line called HL-60. It was the first of the cell lines ever to be established—and there are only a few such cell lines today—of the granulocytic, or myeloid, leukemias. The cell line was established from a patient with a promyelocytic leukemia, and it was done by a postdoctoral fellow of mine named [Dr.] Steven Collins, who is now at the University of Washington in Seattle. We were deliberately trying to grow these cells and one immortalized, and that was unique.

Then Ted Breitman, who, as you will remember, was my mentor, but ironically, when I was a Branch Chief, he worked for two years, in this branch. Breitman discovered that retinoic acid would induce the cell line to differentiate normal cells, stop its growth, and terminally differentiate it. The research was published in a visible journal, but it stayed there rather dormant. Somehow, a decade later, we hear that people are getting dramatic remissions from promyelocytic leukemia with retinoic acid. That is an example maybe of where the transfer of knowledge from the laboratory to the clinic did not work very well. This discovery should have reached the clinic earlier, I think, from the observations we had made and, if I had had a clinic, it would have.

But now I will give you an example of a transfer that did work well. As is described in my book, partly by accident in my laboratory we made the discovery of what today is called interleukin-2. I knew that I was not going to see, nor was anyone with me, the ramifications of this for the clinic. We were using it purely as a practical means for growing human T cells for virological studies and cell biology. But I knew that there were

clinical immunologists in the Cancer Institute [NCI] who could possibly make use of interleukin-2. While the paper was in press, I discussed the results of our work with [Dr. Steven] Steve Rosenberg here at NCI, and with [Dr. Ronald] Ron Herberman, who is now the Director of the Cancer Center at the University of Pittsburgh. Indeed, we began seeing applications of interleukin-2 ultimately into the clinic. That happened very rapidly.

Harden: We will come back to a number of these items.

Gallo: The other question that you did not ask me is what it is like when an M.D. without much—or with little—training in science comes to the NIH. I have described the feeling of awe, but what about when the M.D. gets into a laboratory and does not know anything about equipment and so on? That is an interesting question.

Harden: Let me ask you that question.

Gallo: It gives you a horribly insecure feeling.

Harden: Does it really?

Gallo: Yes. I was very uptight about this, because one of the first things that I saw when I came to NIH was the analytical ultracentrifuge. When I saw it, I said, "I am never going to learn how to use these things." It was a nightmare. All those buttons. I thought, "I will never remember."

Rodrigues: It sounds as though in medical school you had had no contact with such equipment.

Gallo: No, of course not. Analytical ultracentrifuges and things like that? A medical student gets a little exposure to equipment in a biochemistry laboratory, or a physiology laboratory, but not to anything like an analytical ultracentrifuge. I worked in a biology laboratory run by [Dr.] Alan Erslev, where there were rats and mice and partial purification was done of some things, but it was very crude analysis. And bioassays. We had never seen some of the modern equipment that I came into contact with on my first day in the laboratory at NIH.

Harden: Let me ask you then. As a physician, when you were thrust into a basic science laboratory, what was your reaction?

Gallo: My dominant reaction at the beginning, other than the actual curiosity about it all and the desire to do well, was fear. It was a fear that I would not be good enough, and a fear of all this equipment, some of it looking terribly complicated.

I remember a friend from the University of Chicago who was working

with Donald Chudee and who ultimately died of malignant melanoma. One of the first pieces of equipment that he introduced me to, as I was trying to pick up some things for my own research, was the analytical ultracentrifuge. I looked at all the buttons and all the other gizmos on that machine and said, "I am never going to learn how to do modern science, how to use all this equipment." When I first met Dr. [Arnold] Beckman—I had to give a lecture in his honor once about six to eight years ago—I said, "You saved me, because, like other M.D.'s, I could always bank on the Beckman pH meter. I just pressed a button and took a pH." I said, "It gives us a little confidence."

Harden: I remember Dr. James Wyngaarden used to talk about needing either M.D.'s or M.D./Ph.D.'s to provide the interface between the laboratory and the clinic. Maybe you could comment on what a physician brings to laboratory research that a scientist without a medical training does not?

Gallo: What a physician brings to laboratory research that a non-M.D. does not is becoming more blurred now, and I think it is less remarkable. Jim Wyngaarden's book with John Stanbury and Donald Fredrickson, *Genetic Inborn Errors of Metabolism*, which was a book I used to carry around in medical school, was the frontier of medical science at the time. Clearly, in this area, M.D.'s had contributed to, in fact, they had dominated, that kind of research. Before you could get ideas that were important in the field at the time, you had to understand the metabolism and all of the pathophysiology. So, that kind of research was dominated by M.D.'s Not only did they contribute, they were absolutely essential to it. A Ph.D. could not think about such phenomena in the same way at all.

But, as science has progressed, medical science has passed this meta stage, and today there is much more blurring of the difference between an M.D. doing basic research and a Ph.D. doing basic research. Indeed, many Ph.D.'s have now become more sophisticated in their knowledge of medicine. They have gotten more into applications. The new molecular biology brought this about, and I think we are at the stage of being able to apply many of the developments from basic molecular biology to clinical medicine. The Ph.D.'s are well aware of this and their minds are open to learning more in medicine, whereas in the earlier period I think most Ph.D.'s did not want to hear about the medical aspects. Now many cannot get enough of it, and so they start learning more broadly this way.

Meanwhile, an M.D., to survive in basic science, even basic science that has an implication for clinical medicine, has to have the tools of basic science. But I still think that an M.D., in general, has an advantage, with his or her greater breadth in medical biology, and the ability to see things from this perspective. The disadvantage is that sometimes, by temperament and by training, the M.D. has less experience in technology, and less experience in analyzing and criticizing data. I think that those are things that M.D.'s have either to learn on their own or when they are in

training in science. But an M.D. is not trained as fully or as rigorously as a Ph.D.

Harden: I have one or two more background questions before we get into more specific questions. I want to return to the question of the style of managing or leading a laboratory at NIH. I wondered whether there is any person, or idea, that particularly shaped your leadership style. I would also like you to speak about the fact that your laboratory is relatively large compared to many others. Many people prefer smaller, tighter laboratories, but you have a large one. How does this relate to your vision of research?

Gallo: When you ask about style, or about running either a large or a small laboratory, or about which model do I follow in my laboratory, I cannot point to an example. I look up to hundreds of scientists, but I do not think that there is one on whom I have modeled myself. I think perhaps that the person I tend to be most like, although he had a smaller laboratory—not because he wanted a smaller laboratory, but that was the way it was—is Alan Erslev. He was the man who discovered erythropoietin, and I spent some time with him in my summer years in medical school. I would say that his temperament, his personality—probably I was at a stage to be readily influenced by him—may have influenced me more than anything else in these matters. But, in the end, you are a product of your genes and your total environment, of your own will, imagination, and conscience, so all of those characteristics combine to affect your style.

I was also influenced by Sy Perry, by Ted Breitman, and by Sidney Pestka. There was also influence from a distance—it was more than admiration, perhaps the right word is hero worship—from Marshall Nirenberg and a few other people like that. So you do imitate a little. Then I had contact with [Dr. Solomon] Sol Spiegelman outside of NIH. He was an enormous competitor, and certainly increased my competitive instincts and spirit. There was also Bob Huebner with his breadth of vision in virology. You cannot help but be influenced by him when you hear him talk and when you meet him. Things are registering in your neurons—you do not even know it.

Often the other person never gets credit for influencing you because you do not even know it is happening. Sometimes when I have lectured and it has gone well, I hope that some younger people, or maybe even some older people, will be influenced and not even know it. All of the people and experiences combined together to affect me, but I cannot point to one man or woman who overwhelmingly influenced me. I suspect, because of my age and because I think my style is like his, that Alan Erslev may have had the most influence on me. Working with him was my first real job and the first time I had ever done research in a laboratory, so it had a serious effect on me.

Regarding size of laboratory, I think it is a mistake, and sometimes it is not well thought out, for people to argue this way or that way as if they have an answer as to what size is better. A case can be made either way. There is something that the average person, I believe, is always drawn to when you think and talk small. It is "Oh, isn't that nice," whereas, if you are big, it is not so nice. But I believe that is poppycock. People do what they do according to the limits of their talent or what suits them best. There is no science that is better because it is small, or science that is better because it is big. There is no simple answer, actually.

I believe that the greatest scientist in my field in my time, the greatest man I have ever known, was the late [Dr.] Howard Temin. Howard was very different from me, but we were extremely close friends in many different ways, especially during the last decade, and, in that period, especially in the last five years or so. It is interesting, but in time I think we both came to appreciate the opposite point of view. I do not think that Howard, by temperament, by personality, or by his biology, could have run a large laboratory well. Not by brains, but because he just would not have been happy.

I think I would be lonely in a small laboratory, and I think by temperament, by style, and by my need for flexibility, I do better with a significantly sized group. Also, if the research is public-health-related, if it is medically related, you need flexibility. I think it is wrong for an NIH director now, and it was in the past, to try to pose questions about what the size of a laboratory should be. Individuals have different needs according to the nature of the problem they are investigating and according to their own temperament. Let productivity be the judge, and discovery and review. That is the way I see it.

But if you are looking at something such as, let us say, very basic research that has an implication for clinical medicine, sometimes you have to be able to move people from one direction in research to another direction fast. I never can do it by ordering somebody. My style is never to tell a postdoctoral fellow what to do ever. In fact, I have had a few who worked completely outside the main area of research of the laboratory. They did not do well. But the most I can do is to try to influence postdoctoral fellows. The way I could influence them is that I had them talk to everybody in the laboratory, including myself. I would try to convince them that subjects A and B were very important, because I knew where help was needed, but they had to make the decision on what they would work because you take away their excitement if you decide for them. At least that is my opinion.

There are laboratories where research is much more regulated, basic science laboratories and small laboratories, where, when a postdoctoral fellow comes in, he is told exactly what to do. "You are going to make this codon," or something similar, if you were working in Nirenberg's

laboratory at X period of time. But my statement is, "I have a biological problem. Here it is. This is where we are. This is what we are trying to do. These are our long-range ultimate objectives, and these are the people working in the laboratory." I talk to every single one of the postdoctoral fellows. We have staff, section, and unit meetings regularly, and I go to as many of them as I can. I have meetings in my office to try to influence the postdoctoral fellows or the visitor to work in an area that I believe is important and where help is needed. But I think I personally thrive on the flexibility of being able to shift.

We have senior tenured people working independently of me in this laboratory and publishing independently of me. But, in some cases, our origins go back to a similar period of time. For example, let us take [Dr. Genoveffa] Veffa Franchini or [Dr.] Marvin Reitz, who have been here for a long time. They are both section heads. They publish independently. But we overlap in our research, cooperate frequently, and often publish, all three of us, together. We have never had a problem where one or other of them would say, "This is way out. I have no interest in it," because our interests are overlapping. It is natural. It happens without any arguments or fights, and just by virtue of the fact that our interests overlap and there is natural collaboration. I find it enormously beneficial to have that kind of inter-laboratory environment.

If your goal is to work on one enzyme well, you can do good science doing that and it is important. For instance, I know someone in Boston, at Harvard, who worked on an enzyme of herpes virus for his whole career. I do not think it takes a very large laboratory to do that, however. And you are happy. You are happy with your grant of $100,000, or $200,000, and you characterize that enzyme and study its function and biology for a decade or two decades. That can be very good science. It is the kind of science in which I do not think there should be any mistakes, or not too many mistakes. On the other hand, if your goal is—whether it is too large I do not know—to find the cause of a disease, to develop better therapy for that disease, and to understand its pathogenesis to the best of your ability, you had certainly better have a reasonably sizeable laboratory.

I hate the notion that there is a scientific style. I think it is just such hypocrisy and such nonsense. Or this notion of a scientific personality. People think of the more withdrawn person, the shy one, as the scientific personality. If you read books about making the atomic bomb, you find that [Dr.] J. J. Thompson, the man who discovered the electron, fits this personality perfectly. Then, in the same book, you read about Lord Rutherford who is always arguing and telling jokes, sometimes apparently with some color to them. I do not know [Dr. Francis] Crick, but from the stories I hear—I have met him once—if you compare Crick's style to that of J. J. Thompson, they are opposites.

Read [Dr.] François Jacob's book, *The Statue Within*, and you will see all

the different styles of scientists. In this [book] Jacob admits that if it was not for [Dr.] Elie Waldman he would have been trapped many times in being wrong because when he would predict an answer—not to say that it is the method that everybody thinks that you follow.... I found out literally by reading Jacob's book that I do not follow the scientific method most of the time. You go by a certain intuition or a certain belief, or you think that this is how it is going to be and you go after it. Yes, you have to be objective enough to attack your own hypothesis, but you often start with a premise. To return to Jacob, he writes in his book, that often he would get there with one approach, and then he would become bored and go on to something else. But Waldman wanted to see the point that we really wanted proven from twenty directions. Thank God there are different personality styles in science.

Rodrigues: I want to follow up on that. Would you say that your decision to look for a human retrovirus was guided more by analytical thought or was it, as you have indicated, more of an instinctive feeling that this was something to go after?

Gallo: The latter. I think my decision to look for a human retrovirus at the worst time, when people were feeling strongly that one could not exist for multiple reasons, only some of which I put in my book, was certainly more of—I do not know what the right word is—an intuition, a belief, that, yes, there had to be a human retrovirus. The arguments that I was hearing that there was not a human retrovirus had, in my mind—maybe this is the analytical side—little holes in them. There could not be a human retrovirus because so many people had looked for one for so many decades and had not found one. Now, when there is a retrovirus, it replicates a lot and special techniques are not needed to find it. But then my mind was telling me, yes, but it is only the animal model that has been studied, and this was done for the reason that it was easy, because there was a lot of virus. What about the giraffe and the chipmunk in which a retrovirus has not yet been found? Maybe retroviruses are there and causing disease, but their mechanisms of doing so are not by overwhelming viremia. You would never study them because it is difficult and you do not even know that the virus is there. So the models that have been selected are the models in which you know there is a lot of virus. It is easier.

Then the next idea that came along was that in human serum there was the presence of a complement lysing system that could lyse mouse leukemia virus, that could lyse cat leukemia virus, and that could lyse and destroy gibbon ape leukemia virus. Therefore humans cannot be infected by a retrovirus because we have a complement lysing system that destroys retroviruses. Yes, that is true, but only in the few that have been tested. But, I knew that if humans got infected by a retrovirus, it would be one that had evolved to avoid destruction. So, for each argument I was hearing I could think of a counter argument. I had a strong feeling that retroviruses would be found in humans and it became, in time, maybe as

much a belief as it was science, so I guess it was more what you would call an intuitive idea than a careful analysis of the problem.

Harden: We have talked about management styles, but I also want to talk about philosophy and ethics. How does a young investigator pick up the unwritten rules of science in biomedicine? For instance, what decides the pecking order and who gets credit in the authorship of a paper? Is there a clear sense of how this should be arranged or is this a murky area as well?

Gallo: Credit for a laboratory scientific group is, of course, a murky area. There is no rule book. You go by your previous experience, by the mentors that you have had and worked with, and by common sense. I do not think that it is usually a terrible problem. If it were, we would be spending all of our time on such problems, because if you think of the countless papers that are published and the number of scientists today, how often do you hear of a real problem about authorship of a paper? Actually, I should note that you hear about it every time you write a paper. But it only rarely becomes a big issue. Every time you write a paper there is always a little juggling among postdoctoral fellows. Sometimes a technician feels that he or she should be an author on a paper. Then there is the question of the order of the names, who should be first and who should be last. Sometimes it is complicated, but there is no rule book.

I will tell you the rules that we follow in my laboratory. They are not exactly rules; they can easily be broken with discretion. Generally speaking, I have followed a policy that I learned elsewhere at NIH. There is more of a need for the postdoctoral fellows to have publications and not be overwhelmed by every technician in the laboratory. So I have said that before technicians can be listed as authors they have to show some evidence that they have gone beyond the call of duty, such as in their hours of work, or that they have contributed substantively to some new technique, or that they actually know enough to present the paper and have participated in it deeply. In that way a technician can be a co-author of a paper, and, in this laboratory, very frequently is.

Parenthetically, I will tell you that I have often been told by our Associate Scientific Director that I have had more technicians go on to medical school and graduate school than any other laboratory at NCI and maybe even at NIH, percentage-wise. We have a great many who do this. So, something is being done right or wrong. I do not know which it is. Either we are driving them out of the laboratory to make them want to become bigger shots, or we are encouraging them to be interested in medical science.

Many technicians in this laboratory publish because they fulfill one of those three criteria. We just ask them for one. That is my general rule that I tell people when they come in.

After that, I always consider that the person who has done the most primary work, the actual labor—whether a Ph.D., an M.D., a postdoctoral fellow, or the senior investigator—should be the first author on a paper. The last author should be the person who has given the most direction. If it is not at all clear, it is often the laboratory chief. But not always. Sometimes, when it is not clear, a section head is the last author, even though I am involved—I may be in the middle of the authors of a paper—but my involvement was not very great. In time you tend to withdraw from some of those things because you do not need it, and what do you care if you are listed in the middle of the authors of a paper anymore?

So, the leader, the person giving the guidance, the judgment, the criticism, the direction, the head of a section, the head of a unit, or the head of a laboratory is often the last author, but not necessarily. The first author is he or she who has done the most work and who is identified most with the project.

Occasionally authorship becomes a problem. Rarely has there been a shouting match or a real fight, but there have been people who have made significant complaints. Not so long ago a young Ph.D. person was not listed on a paper that she thought she should have been on. A more senior person in this laboratory, the head of a unit, had removed her name from a paper involving gene therapy. This was a big problem. The person came to me. I was right in the middle of the dispute. I was not even associated with the paper, but I had to make extensive inquiries, to find out what happened. The matter is still not settled. Occasionally it can get to that level.

But I can give you other happier examples that are almost funny, one about a paper on which I am the first author and one on which I am the last author.

We discovered a virus from gibbon apes called gibbon ape leukemia virus, a strain that causes T-cell leukemia. Now, Dr. [Thomas] Kawakami in California had found the first virus that caused chronic human myeloid leukemia. We did an immense amount of work with this gibbon ape leukemia virus for one publication. That is, the animal was autopsied, the organs were looked at, and the virus was sought for in every tissue, to find what were the targets—I do not want to bore you—but everything that could possibly be done for one very long paper was done. The paper was for a journal called *Virology*. Everybody did a little of the work. No one could decide who would be the first author. So I volunteered. That was one example where I leapt forward.

Another case, the discovery of HTLV-II, the second human retrovirus, is the opposite of that. The story goes back to 1981. It was in the spring, in Venice, that I heard a talk by [Dr.] David Golde, who was at UCLA [University of California Los Angeles]. He had a new cell line, a CD4+ T-

cell line, which is a mature T-cell line, and it was immortalized. By then, I already had HTLV-I, and from animals I had had some experience with mature T cells. Any cell line that was immortal always had a retrovirus. Golde was using this cell line, and, in fact, his laboratory had had some substantial commercial success with it through a company, Genetics Institute, in Massachusetts, in that it was producing large amounts of lymphokines. At that time, in 1981, it was a hot topic to be able to produce them.

But I got up in Venice and I said, "Look, every HTLV-I transformed T-cell line that I have in the lab, and we can do that every day, produces lymphokines, a high amount of this or that. That is not so interesting to me. But I bet that you have a human retrovirus in your cell line and, since it causes a different disease than the HTLV-I disease, it is probably a new one." So, in fact, I predicted HTLV-II.

We could not get any of that material for a long time. I believe the people at UCLA had made some commitments in their arrangements with the Genetics Institute, but eventually David [Golde] was able to send me just the supernatant, but not the cell line. It is very hard to isolate virus from supernatant alone if it is HTLV because it does not infect. These are viruses that produce only when two cells are brought together. We spent a lot of time, energy, and effort, and eventually were able to isolate virus from the supernatant, and it was HTLV-II.

Now this was another instance where six people contributed substantively to a body of work and no one could decide who would be first author on the paper. This time I did not volunteer. But there was one man, whom I thought had been short-changed on a paper not long before and had taken it in stride, so I said, "He deserves a reward." He became the first author of that paper, a man with an Indian name, [Dr. V.] Kalyanaraman. He did not do any more than any other Tom, Dick, or Harry for that paper intellectually or work-wise, but that is how the decision that he be the first author was made. When nobody came forward as the obvious first author, I, as a laboratory chief, was able to make that suggestion. Everybody agreed to it and thought it was proper and fair.

Most of the time, I think, things work out with respect to authorship, but occasionally they do not work out so easily. But it can be a real dilemma as to who should get the credit. There have been at least two famous cases of complaints by postdoctoral fellows that have led to major awards. Those cases were people complaining that when they were in so-and-so's laboratory, they did the work. It can be argued that whoever was in that slot in that laboratory would have done the work and, if it was not that particular person, it would have been another postdoctoral fellow. The laboratory head, who is leading ongoing research in a certain direction, is, of course, going to get the bulk of the credit and should do so. But if a person came in who was more senior—or even junior—and not only had

the idea, but it was also not in the laboratory chief's line of research, and he or she resisted giving credit, what then? There are cases like that, which people talk about, but it is hard to know what the truth is. In such a situation, the younger person, or the visiting person, has a much stronger case, if their claim is true. This can happen. But generally speaking, if it is your laboratory, and somebody comes into that laboratory and is doing research exactly in the direction of the research you are heading, that person should get credit and probably be first author on a paper about the research. But, clearly, if sooner or later there is an award, it is the laboratory head that gets the award.

There are strange and difficult aspects even in this situation. Let us take the famous story of streptomycin. I read the book this past year. It is a very interesting case because both the people involved were good people. Clearly the author likes [Dr. Selman] Waksman very much, he likes all scientists, because everybody is doing well, but I can think of what a mean reporter might have done with that story. Just read that book again and think of what some reporters would do with that story.

Let us take Albert Schatz—I think I pronounced his name right—this young man who comes to Waksman's laboratory with sugar plums, visions, in his head. Waksman is already well established as a scientist, if not yet as a great scientist. It is Waksman's idea to set up a program to go into the soil and pull out these microbes that release products that have antibacterial effects. He is already in this line of research. That is what his laboratory does.

The graduate student comes and does some work on such material and is frustrated and depressed because his father figure, whom he loved, got all the money and all the credit. When you look at the situation closely, the man had a point. Waksman let him work totally independently, according to the book, and, if I remember correctly, the student was the only one who was exposing himself to the pathogenic strains of *Mycobacterium tuberculosis*. He was working day and night, so much so that somebody found him unconscious in the snow one day.

If you put all those things together and the student is the one who actually isolated streptomycin, it would have been nice if the Nobel Committee could have found a way to give him at least a footnote. It would have been nice if Rutgers could have made him part of the patent.

Schatz corrected this with a lawsuit. That was the sad part of the story because both men apparently liked and respected each other very much. Waksman was shocked about why "Schatz doesn't have respect for me anymore," or why he "has done this to me." "How could he possibly do that, whose career I helped make and whom I recommended and so on." It is possible to identify with either side in this case very easily.

These matters can be murky and complex. I guess your conscience has to be your guide. But the fault is not always with the senior man or woman; sometimes it is over-possessiveness on the part of the younger scientist, thinking that because he or she put things together, he or she did everything. They forget all the contributions that the person above them, let us, for instance, talk about a section head, did for the postdoctoral fellow. These include getting the support, although not loving to do so, doing the grants, setting up the laboratory, setting up the ideas that went into a certain pattern, helping to criticize the results, reviewing the paper, and helping to get it published. All these are things that the person doing some—some, not most—of the work is forgetting, or that they want to forget. There are always two sides to it, but it is a complex matter and there is no way you can write a rule book for it.

Harden: That is worth knowing.

Gallo: That is the truth.

Harden: We want to ask you a series of questions related to the work on interleukin-2 and the first human retrovirus, but, to begin, we would like to set the larger scientific context. You have been involved with the great leaders of the molecular biology revolution—you mentioned Howard Temin a few minutes ago—and with the discovery of reverse transcriptase and so on. Could you make some general comments about the scientific climate for the rise of molecular biology? This would help set the context for discussing your work within it.

Gallo: Are you asking me how did the developments of molecular biology chronologically and productively affect my own research in cancer or in AIDS?

Harden: Yes, and whose great ideas influenced you the most.

Gallo: I am almost afraid to answer such a question because inevitably I will leave out some of the most important people. I may just block them out and not think of them at the moment. Then later I will say, "My God, I forgot the most important person." So, starting with apologies for what I am going to forget and for what I will not have time to mention, the impact of molecular biology on our work and on basic immunology as well was, of course, enormous.

There was no impact early on from molecular biology and not very much from immunology on the discovery of interleukin-2. That is a different story. But for the developments in virology, the development of blood tests, the rapid developments in the understanding of the role of HTLV-I in leukemia and the kind of leukemia it causes, and the role of HIV in AIDS, I cannot give enough credit to the advances in molecular biology and basic immunology.

Interleukin-2 was partly an accident. We were looking for a growth factor. We were not looking for a T-cell growth factor; we were looking for one for myeloid cells. That is pertinent, for reasons that are in my book and that I will not repeat here. We were surveying and screening things very widely. So it was an empirical, intensive, old-fashioned Paul Ehrlich kind of research in the sense that we were screening and testing.

You remember that I told you about the PHA-stimulated lymphocytes that were my control for leukemic cells? Well, once upon a time, in 1971, I think, I happened to look into the medium. We used to throw the medium away. But I began to look into the medium for growth factor and I found [Dr.] Leo Sachs's GM-CSF [granulocyte-macrophage colony-stimulating factor]. This important cytokine, or lymphokine, which makes myeloid cells grow and differentiate, is, in fact, in that conditioned medium. That was one of the first discoveries of a lymphokine. That is, although that molecule was known before, it was one of the first discoveries that T cells were making molecules that were irrelevant for T cells. I remember first telling Leo Sachs, who is from the Weitzman Institute in Israel, about it and that it did not make any sense. Why would T cells be involved with neutrophils? There should be a feedback loop from the neutrophil. That was an empirical observation.

When I was looking for a different growth factor in the middle 1970s, I went back to the T-cell conditioned medium, the medium that we PHA-stimulated T cells for, and made the discovery of T-cell growth factor, now known as interleukin-2. But that discovery would not have been made without the intensity, and the mothering and nurturing aspect, of a woman scientist, [Dr.] Doris Morgan, who had joined me just a short time before. She was doing the experiment as I had outlined it, but I was looking for myeloid cells. She kept coming to me with these lymphocytes and I said, "They will be found to be Epstein-Barr virus transformed B cells. So what?" It happens occasionally, that adults will have immortalized B cells growing, and it is because of EBV [Epstein-Barr virus] infection. Looking at the cells, you cannot tell a T cell from a B cell.

Now comes the next accident. Somebody else in the laboratory, I think it was [Dr. Robert] Bob Gallagher, who is now a clinician, sent the cells to Building 10. I do not know why he did that, but he did. I think it was [Dr.] Ethan Shevach in Building 10 who did the analysis and found that the cells were EBV-negative, immunoglobulin-negative, and therefore they were not B cells. They were lymphocytes. My God, were they T cells? At that time there were very few assays for T cells. T and B cells had only been distinguished a few years earlier, as I recall. But the cells were positive for something called E-rosetting, and they had some other marker that indicated they were T cells. We realized that we had grown T cells for the first time.

I had been, only a few months before, at a lecture by a clinical immunologist from Yale [University], who was talking about how we should not have blood going from older people into younger people because the T cells would be deficient as T cells cannot grow. I mean, with PHA stimulation, one round and it was over. I was sitting there knowing that I was able to grow T cells long-term, but Doris Morgan had to mother those cells very carefully and kept them for a long period of time. Then we realized that there was an active factor as well, that there was a protein. That is the discovery of IL-2. It did not depend on molecular biology or modern immunology. I do not know what it depended upon, probably a series of chance events, a woman who was willing to stay with cells, and some other fortuitous accidents.

But the discovery of HTLV-I, HTLV-II, and HIV, those are different stories. All of those depended powerfully on the developments in molecular biology and immunology. For example, the molecular biology developments that allowed gene cloning, gene sequencing, and learning the structure of the viral genome, were applied to animal retroviruses, so we knew and had a framework for what the genome of a retrovirus was like. Then we discovered HTLV-I, and that made its understanding, its cloning, and the discovery of new genes that were not known in animal retroviruses readily doable. Finding the sequence was due to using molecular hybridization technology in the tumor cells in a clonal fashion that had been all worked out earlier in animal models—the techniques were there—so we could actually just follow them pretty easily. All that we owed to molecular biology.

Who? The names are obvious. The people who developed gene cloning. There were multiple West Coast scientists, particularly at Stanford [University], and people who did the endonuclease restriction patterns, the people who got the Nobel Prize for it, [Dr.] Ham[ilton] Smith, and so on. I do not know where to stop. Reverse transcriptase. Without that I could not have found the virus. So interleukin-2 plus reverse transcriptase, Temin and [David] Baltimore. Without reverse transcriptase, we could not have done a lot more of the gene cloning from the cDNA. We could not quantitate virus readily without the reverse transcriptase assay. We would not have known the virus was there without it, because it was a new virus and there were no probes for it. That assay was absolutely essential. The most anybody had ever done was to see it for an instant. But how to follow it regularly when it is fast, you could just take small aliquots and know when the virus peak was coming out. The AIDS virus comes out with a burst and a peak, and you would miss it if you did not have reverse transcriptase assays. You cannot do EMs [electron micrographs] every 10 minutes.

The blood test technology? Well, we had to mass produce the virus. That was not something from molecular biology or immunology; that was

something from old-fashioned virology, by trial and error. Much of it was developed in Eastern Europe, but there was nothing, as I said, from molecular biology in that.

But, then, in doing the test itself, the ELISA assay [enzyme-linked immunosorbent assay] came out of basic immunology. The Western blot also came out of basic science and we applied it to clinical medicine for the first time. To my knowledge, no one else had done it before, but the test really came out of basic immunology.

I should also have mentioned the people who distinguished T cells from B cells. I forget at the moment who that was. I know it, but I forget.

Harden: There is also the instrumentation that was developed, such as the FACS [fluorescence-activated cell sorter] machine.

Gallo: Yes. I am forgetting the FACS machine, which was obviously helpful in the studies of pathogenesis. Also the people who made the monoclonal antibodies for CD4. Start with that. How did we know to look in CD4 cells if clinicians did not say CD4 went down? How did they know CD4 went down if they did not have an assay for CD4? You do not have an assay unless you have the monoclonals. Who developed those monoclonal antibodies first? There was [Dr. Stuart] Stu Schlossman and the other man with a Chinese name, from the Ortho Company, who did that pioneering work in the mid-1970s, I think, or perhaps the late 1970s. All those monoclonals were critical. Who developed the technique of monoclonal antibodies? You can go back to César Milstein and Georg Kohler. That is the story of science, is it not, scientists standing on the shoulders of earlier scientists.

Rodrigues: To shift gears somewhat, we have been hearing recently about the use of embryonic tissue in research. You touched upon this in your book, saying that you had discussions with Phil Markham in the early 1970s about the ethics of using human embryos in research. Given that there are currently more limitations and restrictions on the use of these types of tissues, if those had been in effect at that time would that have been a stumbling block in your work?

Gallo: Yes. The answer to the question of whether limitations in the use of human embryo tissue would have restricted our research in the early period of time is yes, because the first growth of the myeloid cells—this is not immediately relevant to AIDS—but the first growth of the myeloid or granulocytic cells in our laboratory was based on a growth factor from a first trimester spontaneously aborted human fetus. We do not know the cell that produced it. That is what led me to the discovery of IL-2. We are searching for that factor again, because we could not get more human embryos in time. In fact, we have never found the factor that we were looking for again. We never went back to that question. That is another

story. There is no question that we would not have been able to establish the cell line HL-60 with the restrictions now in effect, which would not have led to the retinoic acid story and the treatment we have today. There is a continuum, but it is not relevant to the issue of AIDS right now, nor for my work in AIDS or leukemia viruses, but it would have been relevant for that retinoic acid story.

Harden: Dr. Harvey Klein told us, when we interviewed him, that in your IL-2 work you made use of the buffy coats that had been spun off from donated blood in the Clinical Center. Would you talk about NIH intramural interaction on this work and the subsequent application of it by Dr. Steven Rosenberg?

Gallo: Yes. The question of intramural interactions in the process of discovery and its applications to clinical medicine are nowhere illustrated better, I think, than at NIH, and especially—well, I do not know if it is especially because it is still going on now—but, anyway, in the period I can recall it was most beneficial for my colleagues, myself, and for our work. It was over and over again. But a good example would be in the interleukin-2 story. Yes. Dr. Harvey Klein was correct. The Clinical Center was routinely sending us the buffy coat material that was not being used for anything else—that was the source of white blood cells. We could set up countless columns and PHA-stimulate them to separate lymphocytes from the granulocytes. If we then take those lymphocytes and PHA-stimulate them, we get the conditioned medium, which is where we found the interleukin-2. We had much contact with the Clinical Center in those earlier years and we obtained a large number of specimens from the Clinical Center. This happens, I might add, much less today. The reason is that there is far more competition for a dwindling number of specimens. If you are not right there in the building, it is much more difficult than it was to obtain them.

At that time, there was almost a search by the Clinical Center for a scientist to be interested in those specimens, more so than today when there is much more competition. As I said, even the Ph.D.'s have become more interested in clinical medicine. But, in addition to the clinicians providing material and patient information for us, and providing the specimens—not just normal buffy coats, but those from patients that they were caring for—they often gave us insights. For instance, in AIDS, knowledge of the CD4 decline came purely from patients. Then there was Steve Rosenberg and [Dr. Ronald] Ron Herberman taking the interleukin-2 story from us to another level by thinking about it clinically, or in mouse experiments that only a clinical immunologist could dream about at that time. There was also our getting the information from Shevach that the cells we had were not B cells. I did not even know that came from him until I was writing my book. I found that out by talking to people in my laboratory. Nobody had ever told me that that was where the information came from. Quite frankly, once I knew that, I thought he should have been

a co-author of the paper. He never made a complaint to me. He probably does not even know. But if he reads my book he will know.

Harden: I have a technical question at this point. Why was the original name "T-cell growth factor" changed to interleukin-2?

Gallo: The original was a better name. It described what the substance did. It made T cells grow. The name was changed by a group of immunologists. I was not invited to the meeting. So, nobody from our laboratory made the change. The immunologists started to use the term interleukins. In fact, I think interleukin-2 appeared earlier than interleukin-1. I think it was the first cytokine that could be defined. But, because in the biology of T cells, it works after an effect of interleukin-1, I believe that is why they called it interleukin-2. In time, the changed name becomes a better name because we have learned that interleukin-2 can do more than grow T cells. But its major biologic activity is still the growth of T cells. It is a descriptive term. Remember, my background was more in cell biology than immunology. Cell biologists use terms such as growth factors, "fibroblast" growth factor, "platelet-derived" growth factor, and it is possible to go on and on. There are many growth factors and that was a common term. What interested me about interleukin-2 was the fact that it was a growth factor. It grew T cells for the first time. What other name would I give it?

But we actually did not call it T-cell growth factor; we called it "T-cell mitogenic factor" in the first paper about it. Then, with the influence of the collaborator, in our second paper we called it TCGF to keep up with the terminology of epidermoid growth factor, platelet-derived growth factor, and fibroblast growth factor. We used the name T-cell growth factor. I think it was roughly two years later, when they wanted to bring some order to the names of lymphokines—what were eventually just called cytokines, in general—and they started using this terminology of "interleukins."

Rodrigues: In talking to other investigators, and based on our own research, one program that came up a number of times when we started talking about the [National] Cancer Institute and its research in the 1960s was the special Virus Cancer Program that Dr. Robert Huebner and Dr. George Todaro ran. You stated in your book that you disagreed with Dr. Huebner's theories about endogenous retroviruses, yet you found the arguments in favor of them highly stimulating. Could you elaborate on this and on the larger value of keen scientific argumentation as a mechanism to stimulate precise thought?

Gallo: The special Virus Cancer Program was actually not headed by Huebner and Todaro, but was first administered by [Dr. Frank] Dick Rauscher and then by [Dr.] John Moloney. But Dr. Huebner and Dr. Todaro were the two most obvious, visible, and famous virologists that were funded by the

program and also, in turn, funded other people by a contract program that was controversial at the time. That led to the Zinder Committee's evaluation of it, but I think that was just the politics of science. There was a lot of money for the special Virus Cancer Program because people had ideas, and Huebner was among those who had the most ideas. Huebner was able to generate enthusiasm and funding for the Cancer Institute when he came in. I think many outside scientists, in time, saw that here was a chance to get good funding and that maybe they too ought to be in cancer research. They were not in favor of continuing the special Virus Cancer Program with its giant contract program.

The special Virus Cancer Program, however, in many respects, made contributions to molecular biology in this country. It contributed to the understanding of a wide variety of viruses and certainly to having reagents available for all kinds of viruses of animals and to some human virus reagents. These were reagents that we capitalized on greatly. How did I know HTLV-I was not an animal virus? We had used reagents from the special Virus Cancer Program to rule this out, before we had characterized HTLV-I chemically and immunologically.

Now, Bob Huebner and George Todaro had a famous theory called the virogene/oncogene theory. It is true that I did not believe in the literal aspects of that theory, and it is true that that theory was not correct. However, the catchy word "oncogene" certainly produced some thoughts about going after a particular gene, or genes. Huebner and Todaro thought it would be one gene originally, or maybe a couple, and it turned out to be a very large number. Their knowledge and ideas that cancer had to involve something in the gene, something in DNA, were already there, so that was not novel. But when you started to speak about it as a specific gene, or a few genes, I think that, in itself, helped to crystallize people's ideas on looking for such genes. But I could not imagine that the theory they were proposing, that virtually all of cancer, if not all cancers, was simply an activation of a set of endogenous retroviruses which included within them an oncogene, was the way cancer developed for many reasons. One reason was that all kinds of activation of endogenous retroviruses in animals were not associated with anything, except publication of papers. You would have it, you did not know what it meant, and there it was.

Also, I was impressed by the lack of evidence, after an intensive amount of work, that such viruses were ever playing a role in cancer. I was more impressed by the people—such as [Dr.] William Jarrett in Glasgow who had discovered feline leukemia virus—who pointed out that, when there was a clearcut viral cause of malignancy in an animal, it came from without.

But Huebner, in retrospect rightly, countered my argument, and not bashfully either, by saying that it was crazy to think of cancer being catching. You will raise the issue of catching. Well, the more I looked,

the more I saw, and the more I thought of models that, increasingly, were showing an acquired virus. Bovine leukemia retrovirus came along in the early 1970s. There was not much virus replication. Maybe humans had the same kind of retrovirus. We did. And it was infective, maybe *in utero*, so it was not seen as a horizontal spread. That happens. We now know that happens in general infection. Quite frankly, I suspect more things happen congenitally in the causes of diseases that we do not have etiologies for right now than we know. I should rephrase that. I think we will see more diseases for which we do not now have etiologies that will be shown ultimately to be due to congenital causes.

Viruses are hard to trace. Epidemiology is almost impossible, especially if there is not high penetration, if you do not get disease every time. Such infections will look like genetic disease as Ludwik Gross pointed out earlier. So, I was impressed that, increasingly, when we knew that a cancer was caused by a virus, it came from without. Maybe it did not replicate much; maybe it infected *in utero*, maybe it came from mother's milk, maybe it made epidemiology complicated, but this was more impressive than the simple expression of an endogenous retrovirus.

However, to repeat, the virogene/oncogene theory crystallized the notion of a gene, or genes, that could be involved in transformation and also that such genes might be captured by a retrovirus—it turns out to be an infecting retrovirus, not an endogenous one. The first identification of an oncogene was in the Rous sarcoma virus and also they picked up such a gene in some of the mouse sarcoma viruses. But they are not just endogenous, then turned on, and cancer follows. The theory was wrong in its detail but it helped—really fermented—many issues, many questions, and promoted much research.

Harden: When you related the story of working on HTLV-I in your book, you talked about losing the cell line that was on a freezer plate and probably carried HTLV-I. This was before the Hershey meeting.

Gallo: Yes. Actually, a slight correction is needed. That mistake is often made because I put them too close in the chapter. It was not HTLV-I, it was just before HTLV-I.

Harden: Yes.

Gallo: It would have been HTLV-I.

Harden: Yes. That was my point. You did not quite know what you had. It probably would have been HTLV-I.

Gallo: Correct.

Harden: We have heard many stories from scientists about freezer failures, yet,

supposedly, the NIH has a very good back-up system to prevent this from happening. What happened in your case?

Gallo: I do not know. We had two disasters from freezer failures. I think we know the origin of one of them. You can get paranoid in the laboratory when things go wrong, and the person closest to the research gets the most paranoid. We found a plug not plugged in, and it was over... That was on one of these occasions. I always get them mixed. But twice we had freezer accidents that were very costly.

In one of the two times it was over a holiday. We came in and found the plug pulled out. People began thinking somebody was sabotaging the experiment, and that sort of thing. But sometimes it is due to the cleaning people. We did not have any back-up in that instance. I do not know what happened. But we lost everything in that freezer.

Harden: What happened at that first meeting at Hershey? You described the disappointment of finding out that your cell line had been contaminated.

Gallo: Right.

Harden: What surprised us was reading that the scientist who reported the contaminations apparently waited to do this in public. Why did they not call you privately? Why were they so bitter? Was this a personal matter or was it related to broader currents in the field of viruses and cancer?

Gallo: No, I do not think it was only personal. It may have been a little of both. Personal, but not really personal, because he thought maybe of the competition and maybe that we were going too far too fast. It would be better to ask someone else who was there that question, like [Dr. Stuart] Stu Aaronson, [Dr. Takis S.] Papas, who is now in Charleston, or [Dr.] Ray Gilden who was there and who participated in that. But, there were plenty of people there. [Dr.] Jeff[rey] Schlom, who is still here, was there. Many people saw that. And [Dr.] Peter Fischinger at Charleston. I think it would be better to ask them. I mean, to put it briefly, it was really long. It was a very difficult time over a two-day period actually.

Yes, I learned from that. I should have given out the samples for everybody to analyze. I went in to the meeting knowing the nature of the problem from our own work, not conclusively—I did not have as much data as they had—but it was already becoming apparent that this was a laboratory contaminant. It was an extraordinary phenomenon. It had never happened before. It seems that there were three viruses, three different monkey viruses, in one culture. This is not something you want to say for the record, but I should say it because it is the truth. It looks awfully suspicious, having three different monkey viruses in one specimen, the thing that would deceive you the most. I spent a long time analyzing what happened. You wonder if somebody was crazy and did it

on purpose.

[Dr.] Robin Weiss came from England to help us in that work and, as previously, there was failure for a month. We could not transmit anything. We had these particles, but we could not transmit them. We asked his help. He came and he could not transmit them either. Then all of a sudden every culture was positive. Then, as the year went by, there were three different monkey viruses in those cultures. Not one, not two, but three. I do not know how this happened, but it was a real disaster at Frederick and it put the field...

Yes, there were. When you asked me about whether it was a personal matter or was it the field, I think there was an aspect in which it was the field, because there was a big push to get rid of the Virus Cancer Program. There was a big push to go completely towards chemical carcinogenesis and just forget all the virus work. So these events coincided. There was a big push to say that there would not be any more retroviruses, and there were already some—I would say in retrospect—silly disasters. There was a virus announcement from the M. D. Anderson Cancer Center and they actually had no data it was a human virus. It was announced as a human virus and it turned out to be a common mouse laboratory virus.

Then Bob Huebner himself had a problem with the so-called RD-114, an endogenous retrovirus of cats. This is an example where knowing something hurt. They put human tumors in a cat, and the tumor came out with virus that was not feline leukemia virus. At that time the concept was ingrained, "one species, one virus, one retrovirus," and that is why you had these type-specific antigens tied to that species in a group across some species. If it was not feline leukemia virus, then it had to be a human virus. In fact, it was a new feline virus; it was the endogenous retrovirus of cats.

I was an author of a publication to say that. I was involved as one of five laboratories. But I really did not feel I had the data. I mean, we all knew and understood. It was not done in a meeting. But in our case, at Hershey, it was done rather dramatically for a whole day and a quarter by one person after another, about ten people in all. So I do not know what drove it the most. I had not had so much success at that time that you could argue that there was somebody jealous or something like that. I do not think there was much to be jealous of. I am not really sure. I want to be honest, so I am going to say I think there was a degree of mean-spiritedness to that show on that day. But I think the story is better told by other people than by me.

Harden: Let me just verify one point again. A number of the people who were criticizing you were NCI contractors and people inside the program?

Gallo: One hundred percent. People at the meeting were either within NCI, in the

special Virus Cancer Program, or contracted to the special Virus Cancer Program. You see, I was not part of the Virus Cancer Program. I was in the Division of Cancer Treatment, of all things, at that time. I am now in the Cancer Etiology Biological Carcinogenesis Area, but then I was not. John Moloney, whom I knew very well, was the head of the Virus Cancer Program, and he gave me extra funds from that program. He transferred money from one division to another because he wondered whether we would maybe find a retrovirus and he thought it appropriate that we would be linked. So we were linked in that way. I suspect that some people in that program were not happy about that, so the attacks came from my competitor and—in his last years of life—friend, Sol Spiegelman from Columbia [University], who was under contract, and one or two people in his laboratory. It came from Ray Gilden, with whom I have worked and collaborated subsequently, who is out at Frederick. He had always been a contractor to Bob Huebner at that time. The criticism came from his associates and from several people who came from George Todaro's branch, in several of the talks, for instance. That was not the end of it, but it was all Virus Cancer Program people. It was relentless, talk after talk.

Rodrigues: I may have missed the answer to this question in your book, but I would like to understand why HTLV-I seems to be isolated in various geographic regions around the world?

Gallo: That is a very good question.

Rodrigues: From my reading, it seems as if the method of transmission of HTLV-I is very similar to that of HIV, and HTLV-I has obviously been around for a long time.

Gallo: HTLV-I is limited geographically much more, at least in causing large numbers of cases in endemic areas, to select parts of the world than HIV has already become. The reason is that HTLV-I, though it is transmitted by precisely the same routes as HIV, is transmitted less efficiently because it does not transmit as an extracellular virion. Remember that I told you we isolated HTLV-II from fluid. That was very difficult. The retroviruses are very poorly infectious as free virions, we believe, because the envelope is very fragile. The envelope falls off very easily. To get HTLV-I to infect, the whole cell and cell-cell contact are needed. So we think that HTLV-I is mostly transmitted as the DNA provirus rather than as a virus. Consequently, it does not mutate as much because that is a more stable form and with fewer rounds of infection, there are fewer chances to mutate. HTLV-I tends to stay within families and be transmitted with particular difficulty. The usual route of transmission is mother to child by milk. Transmission can occur in sex, but it is more difficult. It can occur in blood transfusion.

In studying HTLV-I in endemic areas of the world, it is almost like studying a gene. You can follow migrations of people in ancient times.

[Dr.] Carleton Gajdusek has used it in this way as a tool, in his laboratory, in studying Melanesian people and aboriginal Australians in areas where HTLV-I is endemic. It can be used to follow some of the demography—or whatever the right word is—when you study populations, their movements, their genetics, and so on.

Harden: It is time to shift towards AIDS. I will ask you first the one question that has fascinated me, and actually was raised by Mirko Grmek in his *History of AIDS*, about whether AIDS is an ancient disease or a modern disease. Why did this disease, in your opinion, appear almost immediately after the first human retrovirus had been identified? It almost seems mystical. Grmek suggested that a shift in the balance of diseases in the world occurred: we eradicated smallpox and along came AIDS. But do you have any opinions about the fact that just after the first human retrovirus was identified, we had a retroviral epidemic?

Gallo: AIDS being identified right after the discovery of the first and the second human retroviruses is one heck of an extraordinary phenomenon. All I can say is that it appears to be a coincidence. It has actually misled me. As well as leading me right, it also led me wrong. I put that in my book. For me, AIDS could not conceivably be a different category of a retrovirus. We predicted it was a retrovirus; we were right. We dictated, of course, that it would be in the HTLV family. It was not. So, actually, I think our level of confidence, that we were getting good at predicting, or hypothesizing, probably cost us six months in working on this problem. When I look back on it, we should have had this problem solved in 1982, before the first experiments were even done in France. We started reasonably early, by May of 1982, and should have been done by the fall of 1982, by the end of 1982 at the latest, but we just could not conceive... This is another example of knowing too much, but also not enough. From our experience with HTLV-I and HTLV-II, we thought that we could predict how best to isolate this virus, and we were following our procedures a little too blindly.

But I cannot explain the appearance of AIDS, other than by coincidence. It is possible to say that HIV was identified because of the experience with HTLV-I and II. We were able to think about a retrovirus because we had HTLV-I and II, and we had all this technology, so that made the identification fast. If it were not for that, it might have taken fifteen years. Then you would not say it was close; the events were fifteen years apart. But even that is close. I have to say I think it is a coincidence. I do believe I know the origins of the virus and the origins of the epidemic. I believe I have given the same story since 1984. I do not believe that I have changed my mind in a decade and I do not think any data have appeared that are against what we first said in 1984. But that does not help me in answering the question you raised.

If you want to go through the origin and evolution of HIV and why it

became visible in our time, I think I could explain that, but I cannot explain it as following on the heels of HTLV-I and II.

Harden: I have one related question. We have asked many people, and we would like to have your opinion as well, that if AIDS had struck in 1955, instead of when it did, how would we have responded to it?

Gallo: This is not an opinion. No one can give you a different answer unless they do not have any information—and you would not be talking to anybody like that. Everyone has to give the same answer. Obviously we would have been in a dark box. We would not have known the retrovirus existed for I do not know how long. In 1955 we did not know T cells from B cells. We only knew lymphocytes. So, first of all, we would never have known about a decline in CD4 cells. We would not have known to look at CD4 cells. Secondly, we could not have grown T cells to culture. Thirdly, we had no framework to think about the genome of a retrovirus, so all the advances in understanding of it could not have come.

Could the retrovirus have been isolated? Possibly, but with enormous difficulty, or through some freak accident, because we could not grow T cells. We would not have been able to churn enough in the primary cells to put them in the cell line, as we did, and were only able to do, by late 1983. Very few people had done this at all even then, so I do not see how the virus would have been identified in 1955. Certainly no one would have believed in this kind of virus. They did not even know what this kind of virus was. This was before Ludwik Gross. 1955? All that was known was a chicken sarcoma virus and you did not even have mouse mammary tumor virus by then. You did not have anything. This was just a chicken virus that produced a sarcoma, and these *Visna* kind of viruses were probably not even known then. Maybe they were. I do not know. Certainly nobody on earth knew them but a couple of veterinarians. There is nobody who could answer your question by saying that we would have moved quickly.

Harden: Not even in recognizing epidemiologically that we had a...

Gallo: Sure. If you did the epidemiology before HIV was known to be the cause, you said, "Geez, it's in gay men." Eventually you got some people acquiring it through blood transfusions, so you would have known that it was in gay men and people who had had blood transfusions. I doubt if you would have seen it in heterosexuals. You would never have figured it out. The mother-infant transmission would eventually have become known, that something was being transmitted— the theories that it was non-infectious would have gone away, little-by-little.

When I proposed a retrovirus as the cause of AIDS in 1982, the leading theory for the cause of AIDS, which continued until mid-1983, was that semen was the cause. [Dr.] Gene Shearer of NCI, for example, thought

that. I walked into the Cold Spring Harbor Laboratory Meeting—I think I put this in the book—opened the door, and [Drs.] John Fahey, [Robert] Bob Goode, and several other prominent immunologists were in the front row discussing this theory with great enthusiasm. I remember I did the wrong thing, I chuckled. I was in the back and they all turned around and I said, "Women," They just looked at me and they buzzed to each other and they said, "We are talking about a special form of sex." I looked at them and I said, "Women. If semen was the cause of AIDS, I think women would have got AIDS some time ago." That was the central point. It is good sometimes to think simply and not be too complicated. They had these very complex immunological theories of antibodies to leukocytes and semen that got into the blood through the rectum and then this produced this and it cross-reacted with T cells. It was really an interesting theory. But, I would say, the theories about the cause reverted to infection by the middle of 1983. Most people were accepting the notion of an infectious cause by then. That had not come out before HIV was discovered.

But, look, in March of 1984, NIAID had announced that a fungus was the cause of AIDS. So, there was a great reluctance to think of a retrovirus as the cause of AIDS. My friend [Dr.] Paul Black wrote a letter to *The New England Journal of Medicine* about why it was ridiculous to think that a retrovirus could be the cause of AIDS. After all, we know retroviruses cause cancer. Right?

Rodrigues: I found your comment interesting that looking back on the situation now you feel as though things could have moved faster than they did.

Gallo: No question.

Rodrigues: But yet, from our perspective, it seems as if the NIH's mission and its commitment to long-term research goals were opposed to its capability to deal with rapidly evolving public health problems, as the CDC was able to do. There was not much collaboration with CDC, or historically that was just not going on. NIH did not immediately tackle those sorts of problems.

Gallo: We did not move on them? Look, it has been said that work on AIDS from 1983 to 1985 was the fastest progress in the history of medicine from the inception of a new disease. I agree with that. I think it was enormously rapid progress. Whatever you want to give credit to, molecular biology, immunology, perseverance, NIH funding, hard work, and good ideas all had something to do with it. Whatever that sounds like, that is the truth. It was not some simple thing, and NIH did the bulk of the work. That is, I think, important to understand. Now, if one wants to be self-critical, CDC did the bulk of the epidemiology, but the bulk of the laboratory work in eliminating the virus causes of disease and in finding the right one was done at NIH. The problem was we really did not have a mission, or do not have a mission. I only worked on finding the cause by

accident, by chance, by whim, by feeling that we ought to work on it, that we could, and that maybe it was a good idea.

But, in saying that things could have gone faster, I did not mean NIH could have gone faster, I meant me personally. That does not mean that we were not a little thick in the 1982 period by not being open enough to what was in our... But I really think that, with a little more attention to a couple of details, I could have had the cause of AIDS in hand sooner by a solid year. I was just too much influenced by what I understood from HTLV-I and II. I was waiting for things to be happening in exactly the precise way that they would if it was a member of that family. If you want, I could elaborate on that, but it is a little technical. It is not very difficult technical material, but it may be boring.

Regarding collaboration with CDC, at that time, remember, we had not done research on a public health problem before, at least my laboratory had not. I did not even know what CDC was. I had barely heard of them. I only know that when I wanted donor-recipient matched blood, because clinicians were calling me to say they liked the idea of the retrovirus and wanted us to get an aliquot of the blood, the blood went to CDC and it was not easy to get. Maybe I should not be saying that, but maybe I should, since at least one person from CDC has never been bashful. That was the origin of many problems. We did not get donor-matched blood—it was going elsewhere— that would have helped greatly in determining the etiology data. There was not that kind of cooperation.

Harden: I would like to come back to that in a moment. First, I would like to go through events chronologically. In September 1981, the NCI sponsored a conference on opportunistic infections and Kaposi's sarcoma. This was the first official meeting relating to AIDS. Can you recall that conference and what your thoughts were at that particular time?

Gallo: I get the conferences mixed up.

Harden: This was in 1981, right after the first publication from CDC. The very first one.

Gallo: There were two conferences I went to that affected me. The one that affected me most was when [Dr. James] Jim Curran was provocative, but I do not know which one that was.

Harden: That was later.

Gallo: That was later? I do not think the disease was exciting to me, to be honest, the first time that I heard about it. Yes, it was interesting, but it was small... Early on, things like that were used against everybody in the government, against NIH. It was said that we did not care because it was gay people who had the disease, but this was certainly not true among

scientists. As a matter of fact, I cannot comment beyond my experience in my laboratory, but at NIH itself I never saw anything like that.

The fact was that it was an obscure disease of a small number of people at the time. I was working on leukemia, working—a little bit—at the time on lymphoma, and on aplastic anemia. We already had these viruses in hand. If somebody tells you that a disease called Kaposi's sarcoma, that you had heard about but did not know much about, a rare disease in old Jewish, Italian, and Greek men, has now been found in some gay men in San Francisco and in New York, you say, "Okay, it's interesting." But how many interesting things do we hear about every week in NCI or at NIH? If I started looking in a newspaper and responding to every report about disease incidence, I would be working on a different disease every two weeks. So we did not pay too much attention, although [Dr. Edward] Ed Gelman, who is now the Associate Director of the Vince Lombardi Center, was my postdoctoral fellow then, and we talked a little about it. Ed decided—or we decided together—that he would probe Kaposi DNA for HTLV-I related sequences part-time. This was at the beginning of 1982.

I think the first experiments in this laboratory were in February of 1982. By May of 1982 [Dr. Mikulas] Popovic started doing some culturing, part-time, and by the summer of 1982 so did [Dr.] Prem Sarin.… So I already had two people culturing samples part-time by the spring/summer of 1982. That is a fairly early involvement for the National Cancer Institute, with nobody asking us to do this, and no obligation to continue.

Now, you can argue that maybe NIH should have had some mission of working with CDC at a higher level saying, "Hey, here's a new disease. Let's put some good virologists on to this and some on to that. Let's hear their ideas and support them to do something." But that was never done. Collaboration was all by chance.

Jim Curran of CDC was a positive provocateur. He was saying, "Where the heck are the virologists in this?" He really tried to stimulate some response at NIH, and I listened to him and I got stimulated.

Harden: Dr. Curran was telling us that he thinks your decision to begin work on AIDS was made at the meeting of the National Cancer Advisory Board where you were supposed to be honored for your 1982 Lasker award. He more or less upstaged you because he was giving an epidemiological report on AIDS. But you chatted with him beforehand and became intrigued because the disease involved T cells, on which you were already working. Can you recall that conversation?

Gallo: My memory is funny. I recall more that it was a sunny day, and I recall more the walk back to the laboratory. I recall more that he provoked me, in a way. I was not angry, but it was a little disturbing to be challenged as to "How come there are no virologists involved? Where are the

virologists?" and so on. Curran was certainly thinking of a viral disease as early as anybody in the world, I would say.

I think he was telling me that this was an interesting disease, that it was now more than just a few cases, that it was growing, and that it was important. Things like that I remember. But I probably learned, even though it was published by clinicians, about the CD4 drop from him. And here we were, a laboratory that was doing a lot of work in T-cell biology, and also in virology, and I came back from this meeting and I remember that there was no loss of time in having a discussion with people in the laboratory. I said Curran has got a point. He is an epidemiology fellow from this place that I was now beginning to hear more about, I guess, based somewhere in the South that follows the epidemic. Maybe we should be looking at some of these cases and maybe we should talk to some clinicians. That is how it went.

But he is right. He was the prime mover. If I looked at this historically I think he has been somewhat forgotten, but I would say that he was the prime mover in the entire government. From my perspective, Jim Curran was the prime mover to get people thinking about the disease and doing something.

Rodrigues: We have been going through many records, and one of the groups that seemed to be formed very early on was a Cancer Institute AIDS Task Force, or Advisory Committee.

Gallo: That is right.

Rodrigues: I gather that you chaired this group?

Gallo: Yes. It was the first and only administrative thing I ever had responsibility for in my life, other than this laboratory.

Rodrigues: The part of the story of that group that we could not uncover is actually how it got together, how it was formed. What was the genesis of that group?

Gallo: I think it was just me telling [Dr. Vincent] Vince DeVita that we needed to do something. I needed to get together a band of people. I did not know exactly how to do it. Could I get a little help? Could I get their travel paid for or something like that? I had not had any administrative experience before on something like that. I just said I wanted to do this.

I gathered people together. First, it was my laboratory and then a few people from around the campus, such as Tony Fauci and Sam Broder came. [Dr. Robert] Bob Redfield came over from Walter Reed [Army Medical Center], so it did not cost anything. But then we wanted to bring in a few people, such as [Dr. Myron] Max Essex and later [Dr. William]

Bill Haseltine, and [Dr.] Dani Bolognesi, and some clinicians like [Dr.] Jerry Groopman. As I already said, Redfield came, and [Dr.] Marc Kaplan, people whose thinking I had confidence in, and we hashed over some ideas. Others were Peter Fischinger, and a few people at Frederick. Little by little it grew into a larger group. [Dr.] Wade Parks was another. We just met and debated and thought about what were priorities in trying to figure out what was going on.

That is how the Task Force happened. I think that I just called DeVita up. We had that kind of relationship. That is what is important about access to a Director for the scientific staff. It was not always the same. I had a good relationship with DeVita. I just called him on the phone and said, "I think we should do this," and he agreed.

Rodrigues: How long did that group stay together?

Gallo: The person to get all that history from, she recorded just about everything, is Ann Slisky. Ann Slisky left here to go with her husband to Merck, and then she worked at Rutgers [University] for a while. I think, because of having babies, she is now staying at home. But, if I think again, maybe I have heard that she is back working somewhere. But we have her telephone numbers. She is wonderful. She remembers things. She took notes at every one of those meetings. I do not know where all her notes are right now, but Nancy Miller, who is now one of Fauci's administrative persons and who used to work here, would know where those records are. Ann Slisky's notes would be important, I think. She recorded all the people that were there, and when the meetings were held, and basically what was said and done.

Harden: Was there any formal connection between that group and [Dr. Robert] Bob Gordon's working group out of Building 1?

Gallo: No.

Harden: We have identified a number of different AIDS Working Groups.

Gallo: I did not even know Bob Gordon had one. No. I started the vaccine group too. I thought I would continue with this kind of activity and, immediately, when we knew the cause of AIDS, I went to talk to [Dr.] Hilary Koprowski. We had a meeting in his office with Dani Bolognesi and Peter Fischinger and we tried to do the same thing, have a discussion group for a vaccine. We continued our discussions for a while, but the politics were strongly against it. That group did not last.

Harden: We will stop for now, and continue later. Thank you, Dr. Gallo.

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