



The American Association of Immunologists Oral History Project

Transcription

Dr. Barry R. Bloom, Ph.D.
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Williams: This is an interview with Barry R. Bloom for the American Association of Immunologists Oral History Project. Dr. Bloom is the Joan L. and Julius H. Jacobson Research Professor of Public Health at the Harvard T.H. Chan School of Public Health. He was president of the American Association of Immunologists from 1985 to 1986 and is a Distinguished Fellow of AAI. We are at IMMUNOLOGY2019™ in San Diego, California. Today is Friday, May 10th, 2019, and I am Brien Williams.

Dr. Bloom, I'd like to start by asking you about your family background.

Bloom: My background is pretty much undistinguished other than the fact that everybody in my family, all my uncles and one aunt, were physicians, so it was expected from the day I was born that I was going to be a doctor, I was going to take over my father's practice in ear, nose, and throat and live forever, happily ever after, in Philadelphia. In order to help that mission for me, when I was in high school, they suggested that I apply to a summer program created at the Jackson Memorial Labs in Bar Harbor, Maine, in response to Sputnik. President Kennedy decided they needed to train young people in science. I went up there and had a fantastic experience, did all kinds of transplants in mice as a high school student, and from that time on, I actually knew I wanted to go into science, and if medicine was part of it, that would have been okay, but that wasn't the main interest.

From high school, I went to college at Amherst [College]. I got very interested in doing research in laboratory stuff, an utterly stupid project that was a total failure of irradiating mitochondria and look to see if we could knock out functions, a hopeless project, but great fun for a young college student to think they were doing big-time science.

In a bolt of adolescent rebellion, I decided not to go to medical school, although I had applied and was admitted, and Rockefeller University had started a new program as a graduate university, and this is what I wanted to do. What one of the attractions was was virtually no courses, one introductory course, which was one Nobel laureate after another talking about theoretical physics, quantum physics, everything you could think of, much of which we didn't understand, but we were impressed by the personalities.

Then I decided that immunology was really, really interesting and I went into an immunology lab, and I had five very challenging years working in immunology. The project for my thesis reflects many other projects that I've had, which, in a way, was ending in failure, so I'm a real expert in the nature of failure and the experience of failure as a scientist. The project was to identify molecules that were responsible for transferring cell-mediated immunity. I worked for a famous immunologist named Merrill Chase, and it was Merrill Chase, working in Karl Landsteiner's lab, a Noble laureate, who first showed that you could transfer delayed-type hypersensitivity or contact or tuberculin allergies with white cells

and not with serum. Everybody else had been working on serum and antibodies, and there was something magical about these white cells.

Somewhat before I joined the lab as a graduate student, there were a series of papers published by Professor H. Sherwood Lawrence at New York University, who reported that he could get molecules out of those white cells that, by themselves, could transfer delayed-type hypersensitivity, and as a budding young scientist interested in biochemistry and sorting out molecules, that seemed like a great project. To make a long story short, I learned how to sensitize guinea pigs probably as strongly as anyone has ever done, because that was what my boss really knew how to do. We took out lymph nodes and spleen cells and peritoneal cells and transferred from extremely sensitized animals to naïve recipients, and after three thousand sacrificed animals and five years of my life, not one became positive.

I wrote up my thesis at Rockefeller saying we tried to repeat the human results and they didn't show anything in a population different from humans who had never been prior exposed to tuberculin and other allergens and had some doubts that the human results were right. Wrote up my thesis as a paper for the Rockefeller journal, the *Journal of Experimental Medicine*, where it was promptly rejected by a Nobel laureate on the editorial board who said that they don't publish negative papers in that journal. Undaunted, and by great luck, I was asked to give a talk in Switzerland, and my thesis was published essentially intact in an annual review called *Progress in Allergy* with, I think, six hundred references. Otherwise, I think I would be driving a taxi with no paper and not much to show.

But because of a connection in the laboratory of a prior immunologist who studied in London, I decided I would study, since I had not gotten very far with cell-mediated immunity, I would study antibodies, and I went to work with professor R[odney] R. Porter at St. Mary's Medical School in London. Porter, as you know, won the Nobel Prize for the structure of antibodies, absolutely marvelous guy from Lancashire, very difficult to understand his dialect, but one of the nicest and most generous and intuitively bright individuals I've ever met.

My project was very straightforward. He learned that antibodies had two chains. My project was to find out which chain had the active site. I worked really hard and I worked day and night and I made my own DEAE columns, because in England at that time, you didn't buy kits or columns; you made them. So I learned a lot about how to do science, and to make a long story short, I did not find where the active site of antibodies was, another total failure. It turns out you need both chains, as it turns out, to have an active site. I learned a lot about how to separate chains, and I had a lot of negative results.

I had to return and get a job and I didn't get a huge number of offers, and, for a variety of reasons, wanted to move to New York. My wife was then a student in Asian Studies at Columbia University, so, returned to New York, and the original

job offer was in the neurology department, because they wouldn't hire me in microbiology because I didn't have any papers. But I had an idea of what I wanted to do, which was to go understand a simple question, which is in the cells that infiltrate cell-mediated immune reactions, the tuberculin reaction or an allergic skin reaction, there are two kinds of cells. There's lymphocytes—in those days, we didn't have T cells—and there were macrophages, and the question that was raging was which cell had specificity for antigen.

We worked really hard. My colleague in biology, Boyce Bennett, spent hours with me working how to separate cells, and what we showed is that it was lymphocytes, not macrophages, that had the specificity for antigen, and we showed that using a technique worked out by [Miriam] George and [John H.] Vaughn studied by my colleague at NYU, John [R.] David using cells containing lymphocytes and macrophages allowed to migrate from a capillary tube. If they were in medium, they migrated, and if you added the right antigen, they stopped migrating or they were inhibited from migration, and that was called the migration inhibition assay.

And when we showed that it was the lymphocytes that were inhibiting macrophages and we showed that as few as a half percent of immune lymphocytes would inhibit the migration of the remainder of normal macrophages, we figured out they must be making something and secreting it, so we then looked at what they were making, and that turned out to be very effective. We called it migration inhibitory factor, and that was really the first of the lymphokines that had been discovered and the first non-antibody product of lymphocytes that had been described in the literature. That paper did get published, and in a good journal, so I felt pretty good about that.

Then we did an awful lot of work on the nature of that material and that factor. We showed that if it stuck in the skin, the mix of what we now know are cytokines were able to reproduce cell-mediated immune reaction pathology in guinea pigs, and later, Zanvil Cohn showed that was also true, with Carl Nathan, in putting it into human skin.

So that's how I got started in the business of cell-mediated immunity, a long series of failures, and for reasons not clear, I somehow lucked out at the end.

Williams: So you did this work that you most recently spoke about—was that at [Albert] Einstein [College of Medicine]?

Bloom: That was at Einstein, and after I did that work—actually, before I did that work, the microbiology and immunology department did hire me, and so I was with a wonderful collection of colleagues in an institution that had no endowment, had virtually no money, supported research not at all, but it had the spirit of everyone communicating almost every day in the entire department and in the whole floor working with cell biology. I don't think I've ever seen an environment where

ideas floated around the place between students and fellows and faculty. So with no financial support but a huge amount of intellectual support, we were able to do a lot of interesting things.

Williams: How were you able to buy equipment for your labs and things like that? I mean—

Bloom: We made it. The migration chambers cost about \$2 apiece. They were made out of plastic and they were handmade. This was not high-tech science [laughs], but really very useful. The most expensive was, and remains, animals.

Williams: Mm-hmm, mm-hmm. So you spent time at Rockefeller, you spent time at St. Mary's, and now you're at Einstein.

Bloom: I'm at Harvard.

Williams: I know now you are, but, no, I'm—

Bloom: Ah, in the story, yes.

Williams: In the story.

Bloom: In the narrative.

Williams: Yeah. So can you say some things about your experience of being in those three institutions and how they contrasted?

Bloom: The experience of being a postdoc in England was marvelous and something I would recommend probably to any student. When we do science in the States, we are really privileged to have an enormous number of resources, kits, premade columns, high-technology equipment. At least in those days, in the 1960s, England was a poor country. It had good scientists, but it had very little in the way of material stuff, so you learned to make do, you learned to make things that we would now not think of understanding how does a kit work, how does an ELISA test work. You had to work out the kit yourself. So I appreciated that tremendously.

At Einstein, again, with being totally dependent on NIH [National Institutes of Health] resources, one got used to not living in a world of privilege either, but it was the intellectual environment of close colleagues, all whom worked together, a sensisium of students and fellows, a tremendously great intellectual environment.

The result of the paper on the lymphokines' migration inhibitory factor led to a totally unexpected invitation. When I was a student at Rockefeller, the priority was on basic research. The idea of doing anything applied, i.e., anything useful to anybody on the planet, was something that clearly did not infiltrate much of the thinking. So after we published the *Science* paper on cytokines and which cell had

specificity for antigens, I got an invitation from the World Health Organization from what then was an immunology unit to come to Geneva, explain my work, and the head of that unit had the incredibly naïve view that this was the insight or tool to understand or prevent the most backward of all infectious diseases at the time, which was leprosy. I knew nothing about leprosy. Not many people did know much about leprosy, at least in this country, and nobody, practically, worked on it.

So they arranged a meeting in New Delhi the following year, and there were three outsiders. One had discovered a colony-stimulating factor, which has had a great power in medicine and in the pharmaceutical industry, one discovered that there was a relationship between sickle cell and malaria, and I was the third of that group. Then there were a series of Indian leprologists, and it was all overseen by a young Norwegian named Tore Godal, who later became head of the Special Programme on Tropical Diseases at WHO and the first director of Gavi [Global Alliance for Vaccines and Immunization] and remains an advisor to the government of Norway, an extraordinary connector in science.

It was an extraordinary meeting. It was like a clash of two cultures. We knew nothing about the disease and leprologists knew nothing about science, and the number of questions we could ask was extraordinary that would be easier to answer in leprosy than almost any other condition, because in contrast to TB, as you know, leprosy is caused by a relative of the tubercle bacillus called *Mycobacterium leprae*. We can't study easily what goes on in the human lung. It's very difficult. But leprosy's a skin disease. It rarely disseminates internally, probably because it doesn't grow at high temperatures, and skin is at a lower temperature, about 32 to 34 degrees centigrade. So this is a disease you have to get biopsies from to be able to figure out where the patients are, and that means you can study the development of lesions in a way that is not harmful, actually helpful to patients, and you can follow the course of cell-mediated immunity in an infectious disease, all the activities that are involved in protecting or causing tissue damage.

The second striking thing about this odd disease is that it isn't a single clinical entity; it's a spectrum that correlates perfectly with the immunology. At one end of the spectrum, lepromatous leprosy, the bugs flourish. They grow essentially only in macrophages or Schwann cells around the nerves and they cause nerve damage. In the other end of the pole, there's a massive infiltration of what we would now see as CD4, CD8, and macrophages, and almost no visible bacilli. The macrophages kill of the bacilli, but in the process, they damage the nerves as well. As a consequence, it was an extraordinary, unique opportunity to study the whole range of cellular immune responses from unresponsiveness to too much responsiveness in the context of a human disease. So I have spent a good part of the rest of my life studying immune responses in leprosy, and it has remained, at least for me, a rewarding subject.

The other extraordinary fascination about leprosy is that there's no other disease where the people in the Middle Ages were buried alive, burned at the stake, or thrown out of cities with a bell and candle and left to survive in the deserts on their own. It not only has a history but it has a stigma, and that led me to believe that, yes, I wanted to do basic science, but not just to write papers in *Science*, *Cell*, and *Nature*, but I wanted to do basic science on real diseases with real pathogens, and at the time I started, nobody worked, or virtually nobody, worked on real antigens.

This was the era of reductionist science, people working on model systems, so the major antigen was dinitrophenylated bovine serum albumin [DNP-BSA]. Not aware of anybody dying of bovine serum albumin as a major cause of illness, and here we were working on leprosy bacilli in patients and in animal models, particularly interested in the part of the spectrum where the immune response killed off the bugs but caused tissue damage. That struck me as very odd, and if you think of what tuberculosis is like, it's a massive immune response to wall off the bacilli that causes a hole in the lung and massive tissue damage in the lung. And while we couldn't get access to lungs, the principles, I thought, were likely to be very similar.

The other possibility was that the tissue damage we saw in skin in leprosy might be relevant to autoimmune diseases, and the particular case of interest was the possibility it might be related to multiple sclerosis, where there was infiltration of white cells into the brain with concomitant damage of nerve cells.

So I flew back from India full of enthusiasm, worked with a terrific neuropathologist at Einstein named Henry [M.] Wisniewski, and we did a really simple experiment. We sensitized guinea pigs to tuberculin and then we injected a little tuberculin into the head of some guinea pigs to see what would happen, and what would happen was astonishing: a massive cellular response, a dissociation of the sutures that hold the brain together, and the guinea pigs' heads blew up. So we had created an artificial neurologic autoimmune disease by creating a specific immune response to a foreign antigen in the vicinity of nerve cells in the brain, and the specific response to the tuberculin led to a nonspecific damage of the nerve cells. That's what goes on in tuberculoid leprosy, and, to some extent, that may also go on in some autoimmune diseases elsewhere in the body, and that was called bystander demyelination.

From there, I've worked, for a large part of my career, in collaboration with wonderful students and fellows. The first that I would mention on this occasion is one of my early postdocs, Joanne Flynn, who came with a background in microbiology and is now, I'm proud to say, president of the American Association of Immunologists. It is with enormous pride that I had the privilege of hearing her Presidential Address last night.

As Joanne came to the lab, she was really good at microbiology and genetics and really didn't know anything much about immunology, so we gave her some immunology projects that turned out to be absolutely transformative for the field. It was at that time that knockout mice became available, and so we got mice that had knockouts in gamma interferon [IFN γ] and they died from TB very rapidly, at twenty-one days. We had mice that lacked tumor necrosis factor [(TNF)] and we assumed those mice would not show pathology, but, in fact, they died at the same time as the gamma interferon knockouts. We reconstructed the mice to show that if you triggered both, you need both interferon gamma and TNF to get a protective response in the mice.

Joanne then asked, what about killer cells? And she used mice with knocked out MHC Class 1 and showed that CD8 and cytotoxic cells seemed to be important for protection. Joanne really laid the basis for the fundamental cellular mechanisms of protection, in cellular terms, of how you get protection against leprosy and how you get protection against TB. We still don't know in molecular terms, any more than I did when I was a graduate student, about what molecules are really crucial for assuring protection and being necessary to develop rationally a perfect vaccine.

But to continue my studies in leprosy, I needed to collaborate with someone who was interested in leprosy and interested in TB, but worked on humans, and we don't have a lot of leprosy or TB in Boston, I can assure you, but they do on the West Coast. So I've had a thirty-some year collaboration with Robert Modlin, a superb immunologist, Chairman of Dermatology at UCLA Medical School, and he has always been working on leprosy. I moved him a little bit to work on leprosy and TB, and we reprised the basic experiments to ask the following question, which is what does it take to activate a macrophage to kill TB? TB grows in macrophages and *leprae* grows in macrophages. How do you get the macrophage not to grow the bug and to kill them?

That led to a wonderful set of papers, and we found that there was a mechanism in humans, at least for killing TB in vitro, that depending on products of activated lymphocytes, probably interferon gamma and other cytokines, and it worked by a mechanism totally different than what we found in mice. So in my lab, John Chan and some other students and postdocs showed the major mechanism for killing TB in mice was not oxygen radicals, which is what killed most other bugs at the time, in terms of our knowledge, but it was killed in mice by reactive nitrogen oxides and reactive nitrogen species.

We showed that human macrophage is killed by a different mechanism in vitro, at least, and the mechanism required the engagement of vitamin D. We worked out a completely unique pathway where vitamin D led to the production not of radicals but of a protease or an antimicrobial compound that had the ability to put a hole in the membrane of the very tough membranes of *Mycobacterium tuberculosis* and *leprae*.

To make a very long story short, we've worked out a lot of the mechanisms important for activating macrophages, at least in vitro, to control and kill TB, and we also showed that one of the things that the lymphocytes of the old days, now, CD8 T cells, did is a subset of them could not only kill infected macrophages, they could kill the TB within the macrophages, with work with a young junior faculty named Sam Balin and Robert Modlin's lab at UCLA. That requires a subset of killer cells that is able to put a hole in the membrane of the target cell with perforin and deliver antimicrobial compounds granzyme and granulysin.

So we've put that story together, and in reflection, I think Joanne's work and that of many other fellows in my lab has been extraordinarily informative from mouse to humans. And we're finding now that there are many mechanisms that exist in humans, at least in human macrophages and T cells, that simply do not have genes or do not exist in mice, and so I've really had to shift my focus to collaborative work with Robert on humans to try to find out why things are different in humans and how we really can get what is absolutely essentially to make a better vaccine than BCG [Bacillus Calmette-Guérin], which is the oldest vaccine that we now have for humans, which is protective for kids, not so clear how protective it is in adults, but we don't know why. We don't know why when it works and we don't know why when it doesn't work. So that's the work we are trying to understand, in the process, believing that there are unique aspects of the human immune response, at least regarding cellular immunity, that are just not easily studied in mice.

Williams: How did you come across the effectiveness of vitamin D?

Bloom: This was done by a wonderful postdoc, and it was simply to say if you had activated the macrophages to kill and not the macrophages as controls not to kill, he did an expression of transcription of what genes were turned on in the one and not the other, and usually expect to find between any two different sets of cells 100, 400 differences in genes. He really, after a good informatics analysis, found only two, and one of them was the vitamin D receptor.

From there, it's an interesting story. I got a phone call from Robert Modlin on a Saturday morning and he reported the results of that experiment, and he said, "Bloom, what do you know about vitamin D?"

I said, "Absolutely nothing about vitamin D and its possible role in immunity."

But I did know one thing that I think he had forgotten, and that is the first treatment for TB was sanatoriums, and what you did in sanatoriums is you brought people into the mountains and you sat them all day in what people thought was helpful, fresh air, but in the sunshine. And Vitamin D is made in the body by the sun shining on the skin, converting a precursor to the active form of dihydroxy vitamin D. So it actually made sense that the old guys who ran the

sanatorium didn't know what they were doing, but they may be doing something right.

Williams: Mm-hmm. That's fascinating. So you're talking the science side of all of this. Where does the public health work come into the picture?

Bloom: When I was invited in 1968, I think, to go to India for the first time, it's an experience I really never got over, and I was absolutely motivated to try to use science in some way not to make drugs and vaccines in my lab, but understand the basic science that might allow others to do that, but target it on diseases of the Third World. As a result of that, this wonderful Norwegian who had created the first leprosy center in Africa returned not back to Norway. He was invited to return to WHO and set up a program on leprosy, and he asked me then to join him as an advisor to that program that was called IMMLEP [Steering Committee on the Immunology of Leprosy]. It was funded, tiny amounts of money, by the Norwegian government, and my role was to try to get some of the best scientists in the world to come for nothing to WHO and share their ideas and share something else as well.

It turns out the leprosy bacillus was discovered seven years before Robert Koch discovered the tubercle bacillus. It has never been able to be grown in a test tube. It is a completely genetically degenerate organism. It barely can survive in vivo. It's amazing that it actually causes a disease. Nonetheless, how do you study leprosy if you can't study the bug? It doesn't grow. Except there was a wonderful guy at CDC [Centers for Disease Control and Prevention] named Charles Shepard with another group who showed that it did grow in two animals. It grew in the footpad of mice, which has low body temperature, but you can't get a lot of bacilli out of a footpad in a mouse to study the bug worldwide. But it did grow in a weird animal called the nine-banded armadillo, *Dasypus novemcinctus*, and these are all over the southeast of the United States. They have really crappy immune systems. Because they're encoated with an armor coating, they don't need much of an immune system, and I believe that that's the major reason *M. leprae* grows. They have a low body temperature and they have a lousy immune system.

A contract was let by WHO and enabled two major laboratories studying leprosy to grow enough bacilli in armadillo livers—you could get 10^{10} per gram of tissue. That's a lot of bacilli. WHO organized that, the IMMLEP Committee oversaw that, and the bacilli that were obtained were made available to any scientist in the world qualified to study leprosy, at no cost. While many other infectious diseases, we're worried about giving anything away because they were interested in setting up companies, we were pretty sure there was not going to be a lot of money to be made from a company that worked on leprosy. My lab and another lab elsewhere simultaneously made the first monoclonal antibodies against antigens of *M. leprae* and *M. tuberculosis*. We gave that to WHO, and that was distributed free of charge to everybody that wanted it in the world.

Then I was privileged to meet at WHO with two giants in the field of molecular genetics, Ron [Ronald W.] Davis and Rick [Richard A.] Young, Ron Davis at Stanford, Rick Young at MIT, who had invented the first really useful gene expression system, where you could clone genes into a phage lambda gt10 or gt11 and make foreign proteins from almost anything in *E. coli*, and thus we had the ability to manufacture or at least allow laboratories to make buckets of any TB or *M. leprae* antigen. And in the course of it, the DNA enabled the sequence of *M. leprae* and *M. tuberculosis* to be done. None of this would have gotten done had not a wonderful collection of people outside the field of immunology, outside the field of genetics to be willing to work for WHO for a common purpose to use their skills and knowledge and to make everything that we discovered free and open to anybody in the world.

So I became heavily involved then in WHO activities. I was the first chair of the outside advisory committee called STAC, Scientific and Technical Advisory Committee, to the leprosy program. That led to the creation, since it was such a model program that did the science and gave it all away, that led to the origins of the so-called Tropical Disease Research Programme [Ed. Special Programme for Research and Training in Tropical Diseases (TDR)], which WHO then created for diseases like malaria, leishmaniasis, filariasis, and the hope was that they would also pull scientists from all sorts of fields together to move forward on these now-called neglected tropical diseases. So that has been an extraordinary and wonderful experience.

Then I switched. They created a vaccine program and supported research, and I chaired the committee called IMMTUB, the Immunology of Tuberculosis, and that has spurred on efforts to develop the basic science underlying vaccines. In the last year, we've seen two papers in *The New England Journal [of Medicine]* that indicate there are now hopes for having better ways to immunize people than we've had for the last 100 years. So immunology is really making an impact on two almost totally refractory diseases. TB is now the largest cause of death in the world from any infectious disease, exceeding HIV and malaria for the first time, so this is a serious effort that WHO has inspired and now many labs are contributing to.

Williams: So does this mean that with the vaccine, TB will die out?

Bloom: I don't know when, but I'm pretty confident that—one of the facts, since Jenner in 1796, people forget essentially all vaccines are iterative processes. The first go is never the perfect vaccine, and there is a history of almost all vaccines requiring continuous improvement. So I'm not confident that these two papers are the last word, but if they show, in larger studies, the kind of protection, 50 percent, or for people under twenty-five years of age, one of them showed 84 percent protection, when you're getting 10.7 million new cases a year, I'll take a 50 percent effective vaccine any day. It would be a *huge* impact.

Williams: What about the status of leprosy in today's world?

Bloom: So the status of leprosy is that there was a counterpart committee to IMMLEP which was called TLEP, the mission of which was to develop drugs, and they developed drugs, tested them in mouse models. The situation with leprosy is both interesting and somewhat discouraging. It is interesting because that treatment has been applied to those countries that have a leprosy problem. When I started, there were 12.5 million registered—leprosy is a notifiable disease to all governments. There were 12.5 million patients registered to have leprosy. Assuming that was 50 percent of all the patients, that half were missed, not least because of the stigma and their reluctance to come on, there must have been 25 million at the start of those IMMLEP and TLEP programs. There are now fewer than 800,000 registered new patients. Astonishingly effective impact of drugs, mostly on people who already had the disease. It's a very, very slow disease.

The discouraging part is the incidence rates. The number of new cases has not fallen, and what that means is people are transmitting leprosy before they know they have it and go in for treatment. If they do go in for treatment, they get cured, 90-some percent cures, not a problem. If you put that in the context of TB, the entire global strategy has primarily depended on patients with TB getting sick, and if they're sick, they go to a healthcare place, a provider, and if they're diagnosed, they'll be treated, and if they're treated, they will complete their six months of treatment and be cured.

It's my personal view, not WHO's view, that every one of those premises is mistaken, that there are a fair number of patients who are transmitting leprosy because they're not sick. They have asymptomatic disease, and there's recent evidence that indicates that household contact of patients with TB, healthy contacts, many of them, a third of them, have leprosy bacilli in their sputum and are capable of transmitting infection. Maybe someday they'll get sick, maybe someday their immune response will cure them, but they're sure as hell not going to come in for treatment, which means they're not getting treatment to cure their disease. Once they go, in many countries, for treatment, you have to see three providers who even think about running a diagnostic test to see if they have TB. Well, you can't treat somebody for a disease if you don't know what the disease is. We don't have a good point-of-care diagnostic tool, like HIV in viral assays, so it's a major problem in the field of getting a better diagnostic.

Then many patients feel better in a month, and if they are treated and they stop taking their pills, then they relapse and they take them again and they relapse again, only this time with drug-resistant TB. So the assumption that many people have that we're going to treat our way out of TB and block the incidents of the disease, the new cases, I think is mistaken. It will, over time, maybe by the 2050 deadline, certainly not by the U.N.'s 2030 deadline for the sustainable development goals. We could do better if we had a vaccine and drugs, and that's where the immunology is important.

What's really important there is the only way we have to test a vaccine is very expensive long-term trials in patients at risk for TB, which is a low percentage, even in high-burden areas. What we really need to know is what are the immunologic correlates that guarantee protection. We know for polio if you have neutralizing antibody, you're protected. Done. For hepatitis B, it's exactly the same, and we even know what isotype of antibody guarantees protection. We haven't got a clue what are the essential ingredients to guarantee if you saw these cytokines, these T cells, these NK cells, and innate immune cells, this vaccine worked, this patient is protected. So we have lots of immunology yet to do.

Williams: In 1978, you became a consultant to the White House.

Bloom: [laughs] Yes. I had forgotten that, yes.

Williams: So talk about that.

Bloom: That was an interesting experience. So this was when I guess Jimmy Carter had just become president, and one of the things he wanted to do was to make a major thrust inspired by the CDC person who had been responsible for wiping out smallpox or at least developing the strategy of containment that enabled limited amounts of vaccines to wipe out smallpox, Bill Foege, one of the giants in the field of vaccines and global health. He became head of the Carter Center after he was director of CDC, and that was very high on Jimmy Carter's agenda.

Now, there were a whole slew of people that really knew about global health, but they were all the Kennedy people and the Senator [Jacob] Javits people. They were not Jimmy Carter people. And it turns out the politics were such, they weren't going to be asked for their advice. So how was it possible that they found me, as an assistant professor in the Bronx, to give them advice on how to solve problems of global health? The answer is in my passion for advocating for global health, which was not a popular issue either in science or in the public's mind, I wrote a piece in a journal called the *Hastings Center for Bioethics and Society* [Ed. *Hastings Center Report*], an ethics journal, I think at the time the only ethics journal.

There had been a *huge* debate in newspapers for months about a young woman who had been brain-dead named Karen [A.] Quinlan, and the law had allowed the plug to be pulled for the first time, and her family was at odds. One member of her family wanted, in a humane way, to let her end her life and pulled the plug, and another member of her family wanted her to go on in a state of unresponsiveness forever. I didn't take part in that debate, but given the amount of newsprint and nothing on the millions of people dying of malaria and *Leishmania* and tuberculosis and leprosy, not a word in the papers, I wrote a strong piece critical of the ethicists for not dealing with the global what we now call burden-of-disease problems.

Karen Quinlan was one very tragic case that an enormous amount of time, effort, and money was spent on. We're talking about millions and millions of people for whom simple vaccines, simple diagnostics, simple treatments would make a huge difference in the world, and nobody was paying attention. And somebody paid attention, and that got me, for a year, in the White House working as an advisor to the global health effort in the Carter administration at the beginning of his administration. It was led by an originally English guy named Peter Bourne, very nice man who was the mental health director for the state of Georgia when Jimmy Carter was governor and took what really were snake pits, the worst possible mental health facilities, and made them into medically respectable institutions, a very dedicated guy.

Any rate, we worked for a year. When you were invited to do something like that, you weren't expected to be paid and you weren't. So I flew down about once a week, once every two weeks, and we would meet with various people, and we wrote a magnificent piece. I learned a lot. I thought the problems of global health would be solved or dealt with nationally by physicians, scientists, people who knew what they were doing. I dealt with the Import-Export Bank, I dealt with Department of Treasury, the Department of State, all kinds of financial government institutions. You didn't do global health in the context; you did it in the global political context, and I learned a lot about how you do that. We wrote, I think, a terrific report.

Then something tragic happened. A newspaper article appeared in *The Washington Times*, a conservative Washington newspaper, pointing out that Peter Bourne, the head of this global health effort, had written a prescription for a narcotic for a young woman who was not his patient. He was out of the White House in forty-eight hours. This year's worth of a document ended up not with a press conference but a blue mimeographed cover and was lost to the world. It was released, but with no one knowing what we had done and created, and we had created a major thrust for the United States to take a role of leadership in benefiting the people's health in poor countries.

That was a very tragic outcome, and I learned two things. When I went to Washington and ran around WHO, I thought I would be a pretty good physician politician, if you will, or scholar politician. It's a very nasty business in Washington, which I learned, that hasn't changed for the better, at least in my opinion, since 1968, and I knew I had to be an academic thereafter and have never looked back. [laughter]

Williams: Interesting, interesting. So what were the circumstances that brought about your leaving Einstein and going to Harvard?

Bloom: Adventure. I was happy, doing well, doing what I'd been doing with wonderful students and fellows, getting invited to good meetings and stuff, and out of a clear

blue sky there was the president of Harvard University, called me on the telephone and asked if I would be interested in looking at the job of Dean of the Harvard School of Public Health. I told him I'd never been to a School of Public Health, I'd never studied public health. I was a lab guy. He said, "Well, people have suggested your name, so would you come up and visit?"

So I came up and visited. It was May. He offered me the job, and a month later, I was the Dean of the Harvard School of Public Health. I think the reasoning was it was, for me, a new adventure, and for them, public health was seen as scientifically secondary. It was more back to the old days at Rockefeller, applied research, which was, even at Harvard, given a second-tier category, and I think they really wanted someone who was an active scientist to create active scientists. I took that as both an adventure and a challenge.

Secondly, there was essentially very little or no work on tuberculosis, and all these wonderful fifteen hospitals and research institutes in Boston, TB was just not on the agenda. As I said, there weren't many TB patients to study. So I brought my whole lab up, including Joanne Flynn, and recruited an absolutely super bright guy from Harvard Medical School, Eric Rubin, who's now my department chairman, and he brought Sarah Fortune, one of the brightest people in all of immunology of infectious diseases. And within a year, we had a core mass of really pretty good TB basic research at our little school, the Harvard School of Public Health.

One of the great things that happened there was Sarah is one of the people who just is able to connect with everybody, and she said, "Why are we just having group meetings of our own people here? Why don't we open it up to all of Boston?"

So from my first year there when she set up our Boston TB meeting group, postdocs and students from all over Boston, Tufts, BU, Harvard hospitals, anyone who's doing research on TB is welcome to come, and much of the exciting new work that is done in those labs is presented before papers are written to enable the young people to get criticisms and to bring in outside speakers. So I feel that if I've done nothing else, I've hired wonderful people who've created a community on TB research at the basic level at a really great university.

Williams: Mm-hmm, mm-hmm. I sense the three prongs, sort of, of your career, one being the basic science, and the second would be public health, and the third, maybe not quite different from public health, the advocacy end of things. Tell me what the balance is and where your energy's going.

Bloom: All of the above. A lot depends on the opportunities to be able to make statements. The White House program on global health, even though it didn't reach fruition, brought a large number of people together. As the science evolved and we learned more about the immunology, it turns out you could do basic

science on real bugs. You didn't need DNP bovine serum albumin to study the same basic processes, and that led to the development of a lot of animal models for human disease. The major one that existed when I was a student was actually cancer models, from which tumor necrosis factor and many other basic things were discovered.

But lots of bright scientists working on many different infectious diseases went through the same phase of studying it in animal models and then feeling the need to get into real human studies, and the only way to do that is to reach out for colleagues in developing countries, and to do so in a way that honors and respects them and doesn't exploit them. The history of scientific exploitation of medical people and patients in developing countries in the early part of the last century was shameful, and one of the commitments of the new generation of people in global health is guerilla epidemiology—flying in, taking blood samples, running home, run the assays, and write the paper—that's no longer acceptable.

So I think I've seen, with gratification, the moving from one little committee on leprosy at WHO to a major continuing WHO Programme on Tropical Diseases and a major interest in people doing basic science to relate that basic science to diseases of people in poor countries that don't have the resources of the U.S. or Europe and the NIH, and to do it in a way that is totally collaborative and not exploitative. That's been very gratifying.

Williams: Does that mean that you've set the tone and created a movement that is really gaining ground and acceptance?

Bloom: I would like to believe the movement would have happened certainly without me, but there was a small core of people who legitimized that you could not just do descriptive clinical or therapeutic stuff of these diseases, you could actually use science to understand them, and in understanding them, you would understand regulation of the immune system, how do you get this spectrum in leprosy, how do you deal with an organism like HIV or malaria that is constantly changing its antigens. So I think it's been a mutual relationship of the basic scientists in forming the clinical problems and the clinical problems opening up understanding of basic mechanisms that without them, we would never be able to understand.

Williams: Right, right. Tell me just a little bit about your getting the Gates Foundation grant. How did that come about?

Bloom: More fun than getting a grant. So, I had been involved in not only WHO stuff but lots of committees in the area of global health. There wasn't a huge number of people to pick from if you wanted to have a global health representative, and particularly someone that actually worked in a lab as opposed to collecting epidemiological data. So I was extremely fortunate to be in a position where people found me as sort of the token scientist for a whole slew of policy meetings.

One of those was really very interesting. It occurred, I think around 1990. Every year at the U.N. there's something called the International Year of the something-or-other, and that year was the International Year of the Child [1979], so the U.N. was dedicating itself to doing better for kids. Most vaccines were provided by UNICEF, but UNICEF never did anything in research to make new vaccines. It distributed what existed, and still does.

So this wonderful fellow that did smallpox, Bill Foege, convened a meeting with Jim Grant, who was the head of UNICEF, who was the great social marketer of global health in the world, and the hope was to get more resources for childhood vaccines and maybe even get a little bit of funds to do research to create new vaccines. That failed totally. The board of UNICEF decided it was enough trouble to raise money to get the existing vaccines out. They were not experts in research and they didn't support it.

So not long or many years after that, Bill Gates and Melinda [Gates] had announced they were going to create a foundation, and at the time I'm about to tell you about, they had only one existing program, which is PATH, which was this delivery program for interventions in global health, which still exists and was initially created by the Gates Foundation. They were looking to define what the Gates Foundation would do. The administration, if I remember it correctly, consisted of three people. Bill Gates, Sr., Bill Gates, Jr., and Bill Gates Sr.'s assistant reviewed the program at that time. So they were really looking for what to do.

Because the UNICEF program did not support research on new vaccines and vaccines had sort of been taken for granted, a situation not very different from the current one in the U.S. and Europe now. Vaccine coverage rates dropped from, putatively, 90 percent down to 80 percent. So vaccines were going down, and it was a crisis. So the Rockefeller Foundation ran a series of meetings in Bellagio to figure out what we could do to get new vaccines for which good prospects for development were there and get the existing vaccines we had out. There were five meetings, and it was not very pleasant, because I saw the international bureaucracies competing with each other to get the money and run the whole show. It was not a good thing.

But in the end, it was clear that we hadn't solved the problem, and the head of health at the bank got Bill Gates, Sr. to convene a meeting, having heard of the failure of five meetings of so-called experts and international people, of, "What are we really going to do about vaccines for the poor and disadvantaged of the world?"

So there was a meeting. There was a WHO program that existed that wasn't very effective, called the Childhood Vaccine Initiative [Ed. Children's Vaccine Initiative], and this time, all the same people who had failed five times in a row in Bellagio got their act together because they knew they had to get their act together

for Bill Gates. This was the best shot at getting new resources anyone had ever seen in a lifetime, and they behaved beautifully. The vaccine company people were keen to participate. The head of WHO was there. The head of the World Bank was there. I mean, everybody wanted this to move forward if the Gates Foundation would take the lead. So that was the basic origin of part of the mission of the Gates Foundation to develop new tools for drugs and vaccines.

My role in that was utterly trivial, and the question is should you give the money through WHO to the not very functional global Childhood Vaccine Initiative or create a new entity? They created a new entity, and I was the one, as chair of the meeting, who broke the tie on the name, and that led to the Global Alliance for Vaccines and Immunization, named GAVI. I think if you're going to make a new start, you start with a new name. GAVI has been extraordinarily successful in taking existing vaccines and getting them out into countries where millions and millions of children's lives are saved, so that that meeting at the Gates Foundation was pivotal to creating a new organization that delivered vaccines, but also persuaded the foundation they should be investing in creative research to develop new tools for diseases in poor countries.

They offered some vaccines, research money, and they asked if I would study aerosol vaccines, delivering vaccines for TB to the lungs of mice, which we dutifully did, and the mouse is just not the greatest model for studying immunity in the lung, in my view. So the papers were positive and successful, but haven't led to a new delivery system for humans yet. The studies that Joanne Flynn is doing with Sarah Fortune and her colleagues indicates that, at least in monkeys, aerosol delivery is no better than delivering vaccines the way we traditionally do, intradermally, and there is good reason to believe, from Bob Seder's work, that if you deliver it intravenously, you would get much better results. So I'm not sure if the work we were asked to do, i.e., aerosol delivery, is actually necessary for TB, and we need to get a better way to get systemic immunization.

Williams: Let's turn to the AAI for a moment. You joined in 1967, and what was your motivation for doing that?

Bloom: Oh, you couldn't be an immunologist if you weren't a member of the American Association of Immunology. At that time, there was one meeting of essentially all the biology societies, giant meeting in Atlantic City, where the American Association of Immunologists, pathologists, physiologists, pharmacologists, biochemists all met together, a giant, giant jamboree meeting, and that's how you heard what was—if you were a junior faculty, all the new and exciting things in all these fields. So, yes, you had to join the AAI to know what was happening.

Williams: What was lost when it retracted to just immunology?

Bloom: The meeting got too big. There was a period where that meeting had 15,000 people, and it was too big to do what the societies need to be done, which is to be

able to focus on the next generation and bring them along. I think it was time for them to split off, which they did. FASEB [Federation of American Society for Experimental Biology] is the umbrella organization that still brings the leadership of the professional societies together, scientific societies, but the meetings are separate, and immunology has devolved into—I mean, when I started, there were two things. There was antibodies and delayed hypersensitivity and maybe a little bit on complement. Now it's everything from genes and RNA to tropical diseases, so the meetings have grown to the size of the old meetings of the federation of all the sciences.

Williams: And what led to you becoming the president of AAI in the '85-'86 year?

Bloom: Bad judgment of the membership. [laughter] I have no idea. People were very kind and they put me up, and they put me up with a goal in mind less for what useful I could do for the AAI, but it was the AAI's term next round to be president of the Federation of American Societies for Experimental Biology, FASEB, and a lot of my colleagues wanted an immunologist to be head of FASEB. We were heading into heavy waters in the [Ronald W.] Reagan administration, so I think one of the reasons I got put up was because I had been involved in the White House stuff and knew a little bit about how terrible politics was. Maybe I could protect the society a little bit.

Williams: Mm-hmm. Protect which society, now?

Bloom: Either one. I mean, FASEB is an umbrella group, so if you protect FASEB, you protect the AAI. And that was, for me, an extraordinary experience, because it was a joy being president of the AAI. As always, it has had wonderful staff, so the president didn't have a whole lot to do. A major role was to help suggest names to the Program Committee, who ran the programs and still do beautifully, and to make a visionary speech, which I did, which, unfortunately, does no longer, if ever, exists on PubMed, so nobody can get it, and that had to do with the fact that it had Roman numerals instead of Arabic numerals at the front of the first issue of 1986, and hence it is not retrievable. [Williams laughs.] So I had to ask the historian for the AAI to see my Presidential Address to see what I actually said. Not a bad speech, I have to say.

Williams: What were some of the highlights of that speech?

Bloom: I think there were three things that I tried to put together. One was the burden of disease, how bad things really were in the realm of health in poor countries that most Americans in 1986, most of my colleagues in science really weren't aware of, one in five kids dying and stuff like that in many countries, something about how you could learn from leprosy, as I have described, some really basic immunologic principles that maybe you couldn't learn from mice, and then, thirdly, what the agenda is of what you could do with science, what that would do

to make a difference, if we had drugs and vaccines, how many lives we could save.

That was the thrust of the talk, and I am gratified that we just had a speaker a week ago, now head of Infectious Diseases in [Rutgers] New Jersey Medical School, that said that that talk inspired him to go into immunology of infectious disease. So the platform of the president of the AAI has a great deal more impact both in terms of the presentation to the meeting and also the visionary speech that they give, which is read by junior people and fellows and students and taken as, I hope, a guide of how they can make their impact.

Williams: Mm-hmm. Where was the meeting held during the year of your presidency?

Bloom: I haven't the vaguest recollection. [laughter]

Williams: Okay.

Bloom: And it doesn't say so in the paper, because I read my speech and I tried to figure out where the meeting was held. It's not there. [Ed. St. Louis, MO.]

Williams: Interesting. So tell me about going up to battle with the Reagan administration.

Bloom: So I thought the job at FASEB was a purely honorific perfunctory thing, where we would have the usual dull meetings over coffee and donuts with the heads of all these various societies and they would complain, as usual, about—I think the standard complaint at that era was indirect costs. “We're being ripped off by universities to pay for poets.” That really aggrieved the scientific community, and there was railing against universities and overheads, because we were all losing money from overheads, most of them. But I, because I had been at Einstein and we had to worry a lot about finances, knew that indirect costs were audited retrospectively by the HHS [Department of Health and Human Services], so, no, you didn't put down poets on your overhead and, no, you couldn't take money from your mouse colony to pay poets. But at any rate, that was the sort of interesting dialogue that we had.

As you can tell from my job at Harvard, I believe in institutions. I think science only flourishes if the institutions that enable intellectual life and interactions flourish, so I was sort of an institutionalist on the board, and that became rather important, because President Reagan decided to impound money for NIH that had been voted upon by the House and the Senate; that is, the people's representatives voted on providing money for biomedical research that the President of the United States decided to impound and not spend. That created a crisis because it meant new grants were totally uncertain and continuation of existing grants would be uncertain, and it was an *enormous* trauma to the scientific community at that time.

I received a call, as president of FASEB, from the president of the American Society of Microbiology, who informed me that they had hired a law firm to make the case to get the impounded funds un-impounded; would FASEB agree to join. Well, FASEB could only join if the presidents of the scientific societies agreed, so we had a fascinating meeting, which I shall never forget, where all the society presidents of all the scientific societies were either present or on the telephone, and the issue was should we join the ASM and sue the President of the United States. The law firm was the law firm of Fulbright & Jaworski. [Leon] Jaworski had been the prosecutor, lead prosecutor, in Watergate, pretty effective at dealing with that presidency, and Fulbright was one of the great liberal senators supporting Fulbright international activities, global health, a real hero, and that was the law firm that decided ASM chose to sue the president.

So we debated all afternoon. The FASEB lawyer—they had a consulting lawyer—thought it was unseemly for scientists to sue the President of the United States and strongly recommended that the scientific societies stay out of the politics. I was very unhappy with that. We took some straw votes during the debate and it was pretty split, and so I prolonged the meeting until two of our members on the phone had to leave the conference, and when I knew we had two votes against missing, we took a vote and we agreed to join in the effort and pay, if we had to, to support Fulbright and Jaworski to sue the president.

The lawsuit never took place because once the scientific community joined and once Fulbright & Jaworski were engaged against the president, it was clear the executive branch did not have the power to overrule the will of the Congress, a tension or battle fought then not dissimilar from what can the president do to prevent Congress from doing its work. The outcome of that is the bill was un-impounded, but the delay was so great, we had literally only a very few months to spend a whole year's worth of money because you can't roll over NIH money.

But we won, and that was a very gratifying experience. Again, maybe my little bit of experience at WHO and working in the White House and understanding the battleground of Washington, D.C., may have been helpful in at least joining in a heroic effort to protect science. In that time, we had 6,600 grants from NIH, which was the largest of any time hither to that. So FASEB and the society leaders did very well once we got the money back.

Williams: Mm-hmm, mm-hmm, mm-hmm. So you inferred a parallel with the current scene in some respects. Are you aware of similar efforts today in preserving funding for medical research and whatnot in the [Donald J.] Trump era?

Bloom: I look at my role as, in many of these aspects, of making mischief, and so the current cause of mischief with which I am engaged has to do with the issue of outbreaks of vaccine-preventable diseases in the U.S. As an immunologist, you can't ignore the fact that we have now more measles cases than we've had in the last twenty years. We have a great vaccine for measles, mumps, and German

measles. How is that possible in America? What does it mean to have religious and moral objections to a bloody vaccine that saves lives, and what are we going to do about it?

So in this effort, I can just say that in two editorials, one in *The Washington Post* recently and one in *The Journal of the American Medical Association* hooking up with a major educator, Scott Ratzan, and Larry Gostin, who's head of Health Law at Georgetown, we've put together a set of proposals of how we can tighten up the immunization in this country particularly, to cut out the efforts, quite successful at this point, of the anti-vaccine groups by trying to get the truth about safety and efficacy of vaccines and protect our kids who don't get a vote on this. Kids don't vote on whether they're going to be protected against measles or blindness or deafness or encephalitis or pneumonia; their parents do. And as a result, we've got to get more persuasive in protecting those kids with the tools we have.

So that's the effort that I'm engaged in, and have been quite pleased in the last couple weeks. A whole slew of vaccine experts, which I don't pretend to be, and health law experts and people administrating health programs in universities have started to join and say, "We have to be able to do something to increase the level of awareness and understanding, deal with the social media with misinformation. Vaccines don't cause autism. Vaccines do save lives." That seems to be a battle to take on the politicians right now in—there are only two states that have not had outbreaks, and they're the states you wouldn't guess—Mississippi and West Virginia—who do not have exemptions, other than medical exemptions, for vaccines, and they have had no recent outbreaks. So it's telling you something, and we have to begin to deal with that at a legislative level in states to protect our kids.

Williams: What form is that protest taking?

Bloom: At the moment, writing of editorials. I received a call from the health person in the office of one of our senators asking what could be done in terms of national legislation. One of the things I'd like to be working on—it's complicated—states decide vaccine policy, so it's not easy for governments directly to intervene. On the other hand, there are ways they have of intervening, one of which is to provide money for education, provide money for CDC. When people say they're vaccinated, how do you know their kids are vaccinated? How are we having all these outbreaks? If CDC's average figures is 94 percent covered, all those people who say they have vaccines don't have vaccines, and that requires gumshoe work. People don't appreciate it. A kid with measles that gets hospitalized, which is about one in ten to one in twenty, that's \$125,000 per kid right off the bat just to go into a hospital, hopefully coming out in better shape than they go in. But then there's the whole issue of cost of the public health services, tracking every contact to be sure they're vaccinated. So UCLA has had hundreds of people being tracked to see who has vaccines and who not, and vaccines made available. And two outbreaks in New York. It's thousands of people were screened. There's no

money for that. There is a flat budget for CDC for vaccines, and it doesn't take into account emergencies.

So we are advocating there's a lot that could be done through CDC and through grants to the states to encourage more education in vaccines and, if necessary, to put some constraints to say, "If you don't do better—you're getting a lot of money from the federal government for various aspects of health—we'll put some conditions on them." Whether we'll be successful, I don't know, but it seems like a worthy fight.

Williams: Do you recommend a career in immunology for young people that are considering it at this point?

Bloom: Absolutely. I mean, where can you get a field where you can study fundamental immune responses, regulation of a really complicated system? The dean at Albert Einstein when I was there was a neuroanatomist, and he liked to say to students, who always roared at this, that the brain is the second most important organ of the body, and he never specified what the other organ was. For me, it was always the immune system and lymphoid system, even more complicated, probably, than the brain. The ability to recognize almost any compound, any chemical in the environment that it's never seen before and make a response to it, that's *astounding*, and then to be able to go from very basic mechanisms, from DNA and RNA and all that, to how do you develop a vaccine or a diagnostic that's going to make a difference in the world, I mean, how many fields can one investigator have the freedom to find their niche in any part of a giant scientific spectrum? So it remains even more exciting now than it was when I was a student.

Williams: What do you do for fun besides doing the work that you do?

Bloom: One of the privileges of working in global health and surviving as a scientist in that context and surviving to relatively old age is, on the misguided view that I may have some wisdom not available to younger people, my great fun is being asked to review programs in global health, in immunology, in tuberculosis, in leprosy around the world, and I love travel. I love meeting new people. I love establishing collaborations. I've had a long one when I was in New York and Venezuela for fifteen years. I have Chinese collaborations and committees that I sit on. I get my kicks from sharing what little I can to people who may have not had the good fortune to have the access that I do, and I always come back learning more than I have to contribute. So that's my fun besides hanging out with the smartest group of TB researchers and immunologists in the world every day.

Williams: I thought maybe you were going to say, "I play the cello."

Bloom: I have a harpsichord. I used to play. I haven't practiced in so long, I chose not to mention that, but if I ever retire, which I can't imagine, maybe I'll start to practice more. [laughter]

Williams: Is there anything else you want to say today?

Bloom: No. It's been a pleasure to be interviewed by you, and you're very patient and tolerant. Thank you.

[End of interview]