

Jordan Gutterman, MD

Session 2: April 13, 2012

Chapter 0 **Interview Identifier**

Tacey Ann Rosolowski, PhD

0:00:03.8

Okay. So this is Tacey Ann Rosolowski interviewing Dr. Jordan Gutterman in his office in Research Park at MD Anderson. The date is April 13, 2012, and it is approximately 1:30. This is our second session. So we ended up yesterday's session talking about the great success with interferon and hairy cell leukemia. And you had wanted to—you had just mentioned that you had some dots you wanted to connect to take us to that story forward in a meaningful way.

Jordan Gutterman, MD

0:00:38.0

So I see that Terry Gross is still here. I will stop talking about this. It is so weird. Anyway, so we started—

Tacey Ann Rosolowski, PhD

0:00:49.4

Well, you just promise me that when she interviews you, you will call her Tacey.

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Chapter 7

A: The Researcher

Testing Interferon against Many Cancers

Story Codes

A: The Researcher

C: Personal Reflections, Memories of MD Anderson

C: MD Anderson Past

C: Portraits

C: Discovery, Creativity and Innovation

C: Faith, Values, Beliefs

C: Professional Practice

C: The Professional at Work

A: Overview

A: Definitions, Explanations, Translations

C: Patients

Jordan Gutterman, MD

0:00:53.1

Okay. So when we—so the stuff was cloned, and we started the recombinant. We had to repeat everything with the recombinant that had been done with the natural, and it turned out that virtually everything was the same. The pharmacology—that is, how it behaved in the bloodstream—was the same. The side effects were the same. Although we went to the extremely high doses, in the end the active doses were pretty much the same. So it was a very astonishing thing. And in retrospect, the 1% natural interferon that we were getting from the Finnish Red Cross with buffy coat—the white cells—of normal donors, which probably couldn't be done today because of HIV and so forth—but everything was the same. So the active ingredient was—and the activity was all due to the interferon. The other stuff in there apparently didn't have any—much activity or toxicity, as a matter of fact. And again, we thought that the fever we were seeing was due to the contaminants, so to speak.

[redacted]

So we went through this rather tedious repetition. It was very exciting, but it was also repetitious, and I was eager to move on with other diseases. I mean, I—one thing that characterizes me is I get bored easily. Once I have done something, move on.

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A scientist friend of mine who is a Nobel Laureate—I will leave his name blank for a moment—once talked about another great scientist. This dealt with the Lasker awards. This particular guy is like a golfer—say, Tiger Woods or somebody—who would hit the golf ball 320 yards down the fairway. He moves the ball way down. Now, if it is off to the left by ten yards, or right, it doesn't make that much difference. Then this particular guy said, "Most of us as scientists stand on the green waiting for the ball to get up there." Now, precision becomes important. We have to put it in the little hole. This guy is a guy who can drive that ball and open up something—get that ball so far down that the rest of it is just precision. This particular guy won a Nobel Prize, so he clearly was one of these guys that opened up a field. But I liked the first. I don't—the precision is important, but putting little dots together and finally and so forth, that is—to me, it's not—because I think it takes a certain person, not special necessarily but a certain person to do opening up fields and new ideas and new diseases. But you need the other too. You need both. You know, in a golf game you need to have a putter and a driver and so forth. Okay. So I was eager to try other diseases, but we couldn't do this with the company because it was—we had to repeat everything again. That got to be rather tedious for me because it was obvious for me from the very beginning, having started all this stuff with the interferon for cancer, that it was going to be the same. We weren't going to find anything different.

[redacted]

So the first disease we tried was an uncommon but fatal disease—and at that time, no treatment once it metastasized—kidney cancer—renal cell carcinoma. Not a rare disease, mostly men, and we were really delighted to see in the early '80s—we started this in '81, about the time we started the recombinant—that about 15% , 18%—very small numbers not acceptable to me—however, fifteen did get responses, rarely a complete remission. And we probably did prolong the lives of those patients, although kidney cancer has an unpredictable history, and we reported that. I think it was '82. Mary Lasker was very excited about that. This one I would not have publicized. It was done with the natural interferon, and it came after the wave of publicity in '78 with the ACS report and so forth.

Tacey Ann Rosolowski, PhD

0:05:22.8

Can I interrupt you just for a second?

Jordan Gutterman, MD

0:05:23.5

Yeah.

Tacey Ann Rosolowski, PhD

0:05:23.9

Why did you do that study with natural interferon and not with the recombinant?

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Jordan Gutterman, MD

0:05:27.4

Well, because we were still doing what is called Phase One in pharmacology. We were never—we weren't even close to doing a Phase—so-called Phase Two study. I knew this was going to take time—big time—and I wanted to move on. So we published that, and it was really the first systemic treatment. And still today there aren't really good treatments for kidney cancer. A couple of other drugs have now been approved as well as interferon. Some people do combinations, but that is still an unmet need. That is still a tremendous need. And we don't really understand the very modest activity of interferon, although other compounds aren't too much more active. So that was interesting. But then because of the activity in lymphomas and myeloma—both of which are B-cell diseases—antibody-producing cells—we began to turn our attention towards this rare disease I talked about yesterday called hairy cell leukemia and concurrently with another disease called chronic myeloid leukemia. That, today, is beautifully controlled by Gleevec.

But we had some really, I think, astonishing findings we made in the mid-eighties, which I will now describe. Now, the stimulus for the two blood tumors—hairy cell leukemia and chronic myeloid leukemia—in part came from a conference, our departmental conference led by Dr. [Emil] Freireich, the old DT conference. It was 1982, and we had weekly conferences on all sorts of things. They were blood baths, in a way. I mean, they were pretty chaotic, and a lot of thought was going in, and people said their peace and so forth. It was not easy to present. My colleague Dr. [Jorge] Quesada presented the kidney cancer results.

Now, kidney cancer is a slowly growing cancer, generally, and what is called a more differentiated, let's say, from the acute leukemia or fast-growing tumors. And when we finished that conference, Freireich was really quite excited, and he bellows out in the hallway to me. He says, "Gutterman, come here. It's clear, this working on well-differentiated." That's slowly growing tumors. It works on myeloma and so forth. "Why aren't you doing hairy cell leukemia and chronic myeloid leukemia?" He called it CML. We had been talking about—my group had been talking about this, but I didn't have that—I didn't—I probably lacked a little confidence that it would work. Both were risky. Both were risky because hairy cell leukemia, they didn't have much blood elements. Our stuff, as I said yesterday, lowers blood counts. So I was concerned that we would make—if you are going to make someone who is going to die worse, then I didn't want to do that, of course. And chronic myeloid leukemia, my colleagues all thought we would actually induce what was the fatal step there is—blast crisis. We would take the benign, early phase and stop—even stop that and convert it. In other words, my colleague Dr. Quesada, who worked with me on hairy cell, was all for it. I mean, he was a great supporter, and he was instrumental in this.

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Now, the person doing the CML, Dr. [Moshe] Talpaz, was much more cautious. It took me months to convince him this would be safe to do. And we actually had—I treated a patient with a colleague of mine, Dr. [Kenneth] McCredie, who is long passed away—a colleague of Dr. Freireich's. He treated one patient with CML, chronic myeloid leukemia—I called it CML—and it looked like there was a response. Finally, I convinced Talpaz this was safe to do. I think Dr. Talpaz would maybe have a different story, but that is the story. The story was he was very reluctant. I will tell you about that in a second.

So in late '81, '82, something like that, we began to look at both hairy cell leukemia and CML with the [Dr. Kari] Cantell—the Finnish Red Cross interferon. There was no way Roche or even Schering, if I had been working with Schering, would have taken their precious molecule—first of all, we weren't ready. We had to do all the pharmacology and do all this that and the other. And I was extremely impatient to get going. That was a good decision. That was really, really a good decision. I do credit Freireich, and I have told him this many times that, "You bellowing this out to me in the hallway was the final push to do it." And that was the attitude of DT—of the environment we lived in. I had raised this money with the Interferon Foundation, so we had surplus drug. Now we knew we didn't need any more of the Finnish Red Cross interferon because the recombinant was coming along. So I could afford to be generous now and start looking at other diseases. And that—I don't know where else you could have done that. And I probably need to talk more about the Interferon Foundation. I don't know from the other tape—

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Chapter 8

B: Industry Partnerships

Furthering Research through Partnerships with Drug Companies

Story Codes

A: The Researcher

C: Discovery, Creativity and Innovation

C: Faith, Values, Beliefs

C: Professional Practice

C: The Professional at Work

A: Overview

A: Definitions, Explanations, Translations

A: Character, Values, Beliefs, Talents

B: Industry Partnerships

D: On Pharmaceutical Companies and Industry

Tacey Ann Rosolowski, PhD

0:10:34.1

There wasn't a lot. You talked about the way it was established, Leon Davis and Elaine coming to talk to you, the setting up of it. I think you mentioned a few companies that were involved. But really, that is where the story stopped.

Jordan Gutterman, MD

0:10:49.7

Well, I will fill in the blanks, and there may be other things that one day when I go back and look at my own records I will have every meeting chronicled, whether it was Pennzoil or—and my impressions, the key people. I know some of their names. I have thought about this. So again, I think that we talked about, initially, two sessions—this is going to end up being multiple sessions because I want this right. And I want it documented because the more I talk about it, the more excited—it's very difficult, but I have been able to shut off what is going on now to go back in time, and to take two hours out, once I calm down and get started—you know—once I warm up. So we will go back, because I'm afraid I left out—and I probably will leave out some ideas and names and key people until I look over my oral history. Let's call it that.

Tacey Ann Rosolowski, PhD

0:11:42.6

Well, and the other thing is, remember, that you will get a copy of the transcript and things can be filled in

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Jordan Gutterman, MD

0:11:46.4

Yeah. No, I understand. So I told you yesterday about the hairy cell leukemia and the excitement of seeing the platelets go up first and then the white cells and the red cells and seven of seven patients and the elation of The New England Journal of Medicine and McNeil/Lehrer and people who said that this was all made up and the lady—you know—Dom Perignon with the paper cup. Quite an experience.

[redacted]

And as I said, I told you the story yesterday how the company confirmed all this once we reported all this. And—you know—people ask me a lot, “Well, did you benefit?”—because this eventually became a billion dollar drug—“You must have benefited. You turned over the license. You got it approved.” The answer is no. We never patented anything—ideas or anything. We just—I was just doing clinical medicine. It never even occurred to me.

There are a lot of people including one Nobel Laureate from Biogen who thanked me at dinner one night and said, “Thank you for making me wealthy.” Sometimes it does, but you know what? Life is that way. If somebody asked me—I asked my colleague yesterday, “If somebody offered you a million dollars but you couldn’t do science anymore, what would you choose?” She said, “That is not a complex decision. Forget about it. That is not going to make me happy. I mean, I will take the money, but I wouldn’t change my life for anything.” And I feel the same way. If someone says, “You can’t”—I mean, this is a challenge where I am working now with the plants as this was—as the interferon. This is really a challenge in terms of, again, the politics, the money, and the regulatory. It’s exciting and a challenge, but it’s tough sometimes. And there are some days I’m thinking, “Why am I doing this?” But you couldn’t—there is no price to pay. It’s so interesting.

Anyways, now CML—so chronic myeloid leukemia is—most people who listen to this are aware that in the eighties, again, there was no effective treatment for the disease. In fact, when I first came here, Freireich and his colleagues were using the cell separator to take off the excess white cells. They would give those white cells to patients who needed them, but that’s all they did. There were a couple of drugs then that were used—mustard drugs—that would lower the count. There is one drug called hydroxyurea, which inhibits DNA proliferation—that will lower the count. But none of those drugs—none of the chemotherapy did absolutely anything to the prognosis. The early phase of it is called benign phase—is a single mutation—which at the time we knew a little bit about—a translocation called the Philadelphia chromosome, but then—

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Tacey Ann Rosolowski, PhD

0:17:42.2

I am sorry. I missed that. What is that?

Jordan Gutterman, MD

0:17:43.7

It's called the Philadelphia chromosome.

Tacey Ann Rosolowski, PhD

0:17:45.0

Philadelphia chromosome.

Jordan Gutterman, MD

0:17:47.1

Just the whole research on CML, the molecular aspect is very pioneering. We gave a Lasker Award out for the elucidation of the chromosome defect and the molecular aspects of that. So that was back several years ago. We gave a second award out for the treatment of CML, not with interferon but with this Gleevec, which is so-called targeted therapy. But at the time, in the '80s—'82, '83—there was no treatment. We could lower counts, but there would be nothing for the prognosis. And all the patients—there was an average survival of 3.2 years, and most patients would then convert to what is called blast crisis—acute leukemia—which is sort of like metastatic cancer. And patients would die very quickly.

Tacey Ann Rosolowski, PhD

0:18:39.2

Why is it called blast crisis?

Jordan Gutterman, MD

0:18:41.0

Because they get these undifferentiated cells called myeloblasts. They are blasts. They are very indifferent. They are the first primitive—one of the first primitive cells in the bone marrow. So my colleague Dr. Talpaz started using interferon, and he did a nice job of working on the dose. Right away we began to see the blood counts go down. That was nice. That was prerequisite for anything, but it didn't say anything because a couple of the chemotherapeutic drugs could do the same thing. But the chemotherapy never had a sustained elimination of the leukemia cell—that is, the Philadelphia chromosome—the abnormal chromosome. It was described in Philadelphia by a guy named Peter Nowell many, many years ago.

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Now, I should mention just for completions sake that [E. Donnall] Don Thomas, who was in Seattle at the Fred Hutchinson, won a Nobel Prize for bone marrow transplantation. He showed you could cure people with CML if they had an identical twin and gave their—so you could wipe out the disease if you just blast them with radiation, the chemo, and you could replace the marrow with an identical twin. He won a Nobel Prize for that and related work. But that is pretty rare for an identical twin. Other than that chemo, you might be able to get transient elimination, but they come back. Well, with interferon we saw the blood counts go down.

And I will never forget one day in late '82, about the time we were starting to see these interesting responses in hairy cell—this was heavy time—very exciting time. I see Talpaz way down the hall. He comes up to me—he is an Israeli guy—and he said, “Jordan, we got a bone marrow report back on a patient who has 50% reduction in the Philadelphia chromosome.” I said, “Really?” I said—same thing I had said with the hairy cell—“Well, I’ve got to repeat that, first of all. Is this an artifact?” But—you know—we thought maybe it was a transient thing. I mean, I was very excited. The patient would come back a month later, and now it was down to fifteen percent or something. This thing was disappearing. And then we did it a second time.

Now, it wasn’t like hairy cell, where every patient or virtually every patient responded. Getting some suppression occurred in maybe 30%, 40% of patients. Getting a complete suppression was uncommon, but still it was amazing. I mean, we saw a complete elimination in perhaps 8% to 10%. But nonetheless—and then we worked—some patients stayed on the interferon. Interferon you cannot stay on indefinitely because you are just fatigued all the time. So we had to work out various aspects. Some people we would stop. Sometimes the disease did not come back at all. So I would say that I don’t have the precise figure today, but I would say we probably, with interferon alone, cured a small fraction of these patients, maybe 5% to 8%. But nonetheless, for a disease that has, say, 4000 or 5000 patients, that is still some souls. That is some people. I personally think this has been missed—the importance of interferon—because it was the first demonstration outside of the bone marrow transplant in identical twins that with a compound—with a drug—you could—in some patients—not even close to 100%—nothing like hairy cell—you could get selective suppression of the malignant clone of cells and get, at the same time—which really shocked me—restoration of the normal cells. So while you are suppressing the malignant stuff, you are allowing the normal cells to come back. We saw the same thing with hairy cell leukemia, but we didn’t have that type of marker. We eliminated the hairy cells. And in contrast to normal people—I mean—excuse me—people that didn’t have blood cancers, where we lowered the blood count, in this case, the normal cells were able to restore themselves.

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So I think from a scientific standpoint—but the one criticism of the work—and I agree with it—is scientists need to be precise. Nobody understood exactly why this was going on, and it's hard to build on that. It's wonderful. And we now have a new treatment. The first report was 1983, in Blood. And then in '86, the year interferon was approved for hairy cell leukemia, we had a major paper in The New England Journal of Medicine where we showed this Philadelphia chromosome, and it got a lot of attention because this was historical. Ten years later, a drug called Gleevec was designed by Novartis with the push of Brian Druker, who shared a Lasker Award for this. So I was involved with both of those awards along with two other guys, where they targeted the Philadelphia chromosome.

Ninety percent of patients achieve remissions. Most of them have suppression—partial and usually complete—of the Philadelphia chromosome, and it's an oral drug. They stay on it indefinitely. And it is a controlled disease in the vast majority of patients. The only negative about Gleevec is it probably cures very few people. They just have to stay on it. But how many diseases do we cure outside of infectious diseases? We don't cure diabetes, but we control it with insulin. There are a lot of diseases like that. So this was a major advance, and that is why Druker shared the—and he deserves the most credit. A guy named [Charles] Sawyers shared that with him. He worked on resistance.

Studies are going on with interferon plus Gleevec. I'm not aware—there has been some inconsistency whether the two are additive in any way, because most cancers—like most complex microbial infections, like TB and so forth, you best use combinations of drugs to prevent emergence of resistant cells. But nonetheless, interferon was a major advance. And it took another ten years before the real major advance. But still, I am very proud of that work. And it really showed you how to get selected suppression. I still think there is probably going to be a role for interferon in CML. Hairy cell, too, was supplanted by drugs, which were easier to give, less toxic. It's still used today, but that's okay. I mean, it would be like—I'm not equating it necessarily—but it would be like sulfa. Most people think penicillin was the first antibiotic. Sulfa drugs were. They are still used too. But penicillin was better, and now we have some highly effective—obviously tons of effective—so interferon is used in CML. It is used in hairy cell, but probably better—not probably but definitely—better in less toxic compounds are being used. But now hairy cell was on the map, and CML is on the map. So that is pretty exciting stuff.

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I haven't been staying on top of those fields, although there are advances in these diseases and others. I noticed just yesterday there has been a resurgence of interest in interferon as an immunotherapy that is enhancing the immune system, so I think we are going to see much more use of interferon. People have asked me, "Is it used in all these diseases that you started?" The answer is not as much. And I think part of the reason is there is no champion like myself, I think. I mean, I'll push if I think there is a real reason to use it. I don't know. I've been a little disappointed that the—for a while, the momentum of interferon—people, again, were doing the cleanup, you know? They are putting the ball in the green. But I think there's going to be a resurgence of interferon in terms of the cancer—in terms of figuring out newer mechanisms. I think if you could figure out precisely how it works it would help, particularly today with targeted therapy. Oncology community, patients, doctors, FDA, and so forth want to understand how this drug works. I don't think we can get along any further by just giving a drug that works without understanding. I think what is most important is that it works. If you understand it, that is better.

But as far as being on top of the list of drugs, I think people want to understand how they work first, and I agree with that. If you take antihypertensive, if you take anticholesterol, we understand how those drugs work, and they are extremely effective. So if there is one major criticism I have—and I got out of the field because I said I like to open things up, and then after it was approved—and I will come back to this—I learned a lot about marketing and how this all does. We made a few more advances, but then there were so many people in the field; I really wanted to start a whole new thing. I thought we needed different answers. I haven't stayed on top of this very much. And who knows what will happen, because what I am doing now—which we won't talk about today—may have an interface with interferon and perhaps other things.

Okay, so in 1986, as I mentioned yesterday, in June—mid-June—the drug was approved by the FDA. It was just a little notice in the Wall Street Journal, I remember, that it had been approved. But this was anticlimactic. We knew it was going to be approved. I mentioned that in a few months before we had gone—I had gone with Roche to the FDA, and Schering went. They were back-to-back presentations. It was almost a forgone conclusion, I mean, you could not deny the use of interferon for hairy cell leukemia. It was an incurable disease at the time—nothing worked—and 90% of patients would benefit. So it was a slam dunk, so to speak.

Tacey Ann Rosolowski, PhD

0:28:53.2

Can I ask you just a quick clarification question? You have been mentioning Roche and Schering, and were you talking about agents of those companies or actually people by those names?

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Jordan Gutterman, MD

0:29:05.3

No, Roche is Hoffmann-La Roche, a Swiss company.

Tacey Ann Rosolowski, PhD

0:29:08.6

Right.

Jordan Gutterman, MD

0:29:09.0

And Schering is Schering-Plough.

Tacey Ann Rosolowski, PhD

0:29:10.5

Okay, so there were agents of those that were coming to these meetings with you?

Jordan Gutterman, MD

0:29:13.5

Yeah.

Tacey Ann Rosolowski, PhD

0:29:14.0

Okay. I just wondered if it was actually a person who was named that. Okay.

Jordan Gutterman, MD

0:29:18.1

No, no. I had mentioned yesterday a couple of the names that were at the FDA meeting. I worked with Hoffmann-La Roche because I had gone there. It should be on the earlier tapes with Lesley [Brunet]. I went there June 15, 1978. I think it was the same trip, if I am not mistaken—same week as—no, no. It was June 15, 1978. I had gone there, I believe—something like that—after we had seen these responses in breast cancer with the natural interferon to meet with Dr. [John] Burns and Dr. [Sidney] Pestka, who eventually played a key role in cloning interferon-alpha for Roche—Roche Genentech. And there was another group, Biogen—a Swiss company—a Dr. [Charles] Weissmann—that was working. And Schering-Plough licensed that compound. Those were very exciting days. I think I described them on the previous day, but if I see it is left out, we will fill all that stuff in. Those were interesting days. And I, for the first time again, understand the pharmaceutical industry and also the nascence of the biotech industry—you know—Genentech and Roche—because I went with Mary Lasker in the late seventies to Genentech. Then [Robert] Bob Swanson—and my guess is that that is on the previous tapes, but if they are not, that is going to be important to go through.

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Tacey Ann Rosolowski, PhD

0:30:41.4

I don't remember the detail that you gave, but you did talk about the beginnings of the biotech industry with Lesley.

Jordan Gutterman, MD

0:30:49.4

Yeah. So I think we are going to hold that. I don't want to be redundant.

Tacey Ann Rosolowski, PhD

0:30:51.3

Yeah, until we have a little bit more—yes

Jordan Gutterman, MD

0:30:55.0

Okay, so interferon gets approved in June of '86, and we're starting to think about branching out to other, as I called them, cytokines. And the big push in the mid-eighties was a discovery originally from a guy named Don Metcalf who won a Lasker Award for this work on growth factors—proteins—that allowed red cells to mature. One of the first ones was erythropoietin, EPO, which stimulates red cell production. It is produced in the kidney. A company called Amgen developed that for anemias, especially with dialysis. There was a compound called GM-CSF. That was developed by two or three companies—Schering-Plough and a new biotech company at the time called Immunex, which was started in 1980 in Seattle.

Tacey Ann Rosolowski, PhD

0:31:59.8

What was the name of that drug you mentioned before?

Jordan Gutterman, MD

0:32:02.1

EPO?

Tacey Ann Rosolowski, PhD

0:32:03.5

No, the one after that.

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Jordan Gutterman, MD

0:32:04.7

GM-CSF.

Tacey Ann Rosolowski, PhD

0:32:05.4

Yes, GM-CSF. Thank you.

Jordan Gutterman, MD

0:32:08.9

That stimulates white cell formation. The M is for macrophages because it kind of activates the immune system. The connection with Immunex is important, but it becomes particularly important in the story of the nineties and the 2000s. I won't get into that, but that becomes—my connection with Immunex really has a huge impact on what I'm going to tell you at some point.

So I met the two principles of Immunex, the company that was started by two immunologists out of the Fred Hutchison Cancer Center—Steve Gillis and Chris Henney. They started this in 1980. It was interesting, actually. Again, being here at—and I don't want to lose track of talking about MD Anderson, but this was all going on as I was a professor here and responsible for seeing patients and doing this research and then meeting these people, because I could see a lot of the drugs were being produced not by government but by biotech and the big pharma. So we had to figure out a way of working with these people, and it's still complex—conflicts of interest, money gets involved, and so forth. But I was approached by Dr. Henney and Dr. Gillis. They knew about the work with interferon. Could we—would we be the first to do a growth factor in human patients to restore blood counts in people who get chemotherapy primarily? That was the whole idea. And they produced a—called GM-CSF. I think their trade name was Leukine. I hope I am not making a mistake here—L-E-U-K-I-N-E.

Anyway, in '86 we started the first study, as far as I know, as I remember—I think it was around June—around the time interferon was approved—the first study in giving a chemical—a protein GM-CSF to restore blood counts, white cells for sure, and maybe platelets in people getting chemotherapy or in people with diseases that had bone marrow failure, like a disease called aplastic anemia or a complex called MDS, or myelodysplastic syndrome. These people have very low blood counts, and they die of either infection—they either convert to leukemia or if not, before then, then they die of infection or bleeding. It was a very exciting time again, and we were not surprised to see the blood counts go up.

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I worked with a woman who originally—her family is from India—Dr. [Saroj] Vadhan-Raj, who is still at MD Anderson. She did the first studies with me. We had two New England Journal papers, which says we did some really interesting things. We treated patients—we restored some of the blood counts of this disease called aplastic anemia and also, surprisingly, in this myelodysplastic, which is a pre-leukemic syndrome. The best of the group is a drug called G-CSF, and the trade name is Neupogen. This was produced by Amgen. And that came just a little bit later. That has ended up being the drug because it has fewer side effects than the GM-CSF. It made Amgen.

Those two drugs made Amgen. EPO for the red cells—you know—it is a huge, multi-billion dollar company. I remember going to Amgen in '83 because they were interested in interferon because we had just—we hadn't reported the hairy cell yet. I thought this company has no clue what they are doing. They were an early recombinant cloning company. This was '83. I never—I missed it completely. I missed it completely that they would become a multi-billion dollar company. The head of that company, George Rathmann, was a great visionary. He was determined to use erythropoietin to restore red cells in dialysis patients. And Genentech, I understand, turned that opportunity down. That became a big seller. And it became—it is a whole story unto itself. Then they licensed a drug called pluripotin from Sloan-Kettering for, I think, around \$40 million, and that became a multi-billion dollar drug. It definitely supplanted the GM-CSF that we were working on.

But it wasn't an area that I was passionate about. It was interesting. We got some nice science. We did a lot of studies. But it wasn't—you know—it maybe allowed for more chemo patients to recover more quickly or certainly not die of infections. So it definitely has a clinical use. But it wasn't my major interest in research. So we did a lot of studies. We published a lot of papers, but—

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Chapter 9

B: Industry Partnerships

Research Money: The Economics of Drug Companies; Philanthropy

Story Codes

A: The Researcher

B: Industry Partnerships

D: On Pharmaceutical Companies and Industry

D: Business of Research

D: Fiscal Realities in Healthcare

D: On Philanthropy and Volunteerism

D: On Research and Researchers

D: Understanding Cancer, the History of Science, Cancer Research

D: The History of Health Care, Patient Care

C: Professional Practice

C: The Professional at Work

A: Overview

A: Definitions, Explanations, Translations

A: Character, Values, Beliefs, Talents

A: Personal Background

Tacey Ann Rosolowski, PhD

0:37:08.2

Did you have any big shifts in perspective? I mean, because this was really approaching research from an entirely different way—going through drug companies, basically.

Jordan Gutterman, MD

0:37:19.5

Yes. Yeah.

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Tacey Ann Rosolowski, PhD

0:37:20.6

So what changed, and how did that enhance your outlook? Or what did you learn from the experience?

Jordan Gutterman, MD

0:37:29.3

Well, now—okay, so that is going to come up later. It's a very insightful question. It's a very good question. To go back for just a second—and I'm really going back because you've opened up an interesting question. I haven't talked about this today, for sure, or yesterday, and I don't think with Lesley even. But actually, the first compound I worked with was BCG, and I may have talked about that.

Tacey Ann Rosolowski, PhD

0:37:55.3

You talked about it some in the first—

Jordan Gutterman, MD

0:37:57.3

This was back in 1971, 1972. It was an immune stimulant. And now immunotherapy is becoming a field. It is used to—it is approved for bladder cancer. And we bought that stuff. Well, yeah, I think we mostly bought it. I'm not going to go into that. I didn't see a great future for this. And then when I became familiar with interferon and went to that conference in '75 when no one would go here, I knew we had something we could measure, we could quantitate it, it was a protein, and I could see that cloning genes could be the future here. That was much more exciting than using some old vaccine that had been around. Also, I thought the science of interferon was much more interesting.

The first study with interferon, I was not with a drug company. We bought the stuff. We bought it. We actually—you know—and Mary Lasker gave that first million. Then I had to raise the money. So that was the first track I took after BCG, treading new ground, all the time not knowing this. And I do need to talk in more detail about the Interferon Foundation, because Mary Lasker turned the switch on. Without that, nothing would have happened. I mean, nobody gets really the right credit when they talk about interferon today because the history of all this stuff. I find that people forget history, and I think history is important. I think what we are doing, what you are doing with the library, what MD Anderson is doing—you need this for younger people to learn this. And a lot of it is on the fly. I mean, I—so that was not with a drug company.

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So that is how I started. Then I realized we can't keep buying this stuff, and there is a new historical technology—recombinant DNA. By the way, one of the co-discoverers—inventors of recombinant DNA, Stanley Cohen, was my resident when I was an intern at Duke. I still see him. He is on our Lasker jury. So he and Herb Boyer, who was the co-founder of Genentech started this whole thing in '73, just at the time when we were thinking about all this stuff. It's really exciting.

So I realized we had to work with companies. You can't make—you can't—I mean—the government is not going to do it. MD Anderson is not going to do it. You can't afford it. So it was a natural thing to drift into. When I went to Roche in '78 with Mary Lasker and her nephew Jim Fordyce and her sister Alice Fordyce, I was overwhelmed by the technology of the Roche Institute. Now, that was an unusual place because they had set up a research institute on the campus of Hoffman-La Roche in Nutley, New Jersey. I was overwhelmed by the columns, by the science. They were trying to clone genes. I mean, this was science fiction. This was total science fiction. I mean, I could have flown back on my own I was so excited to see the power of working with intelligent scientists and business people in the pharmaceutical industry. That has changed a lot. Not completely. We will come back to that later. We have had to adjust to the times.

Then simultaneously I heard about Genentech that was started April 7, 1976, which is ironically—the anniversary was just last Saturday. It's also the day—it was two years to the date that my father died that Genentech was formed. And I learned about that. Then Jim Fordyce, Mary Lasker's nephew was—I'm answering your question about big pharma and biotech. Jim Fordyce shared an office with Bob Swanson, the founder and CEO of Genentech who unfortunately died of a brain cancer. Mary, Jim, and I went out there to meet Swanson in '78 or '79, in San Francisco. I remember going to another company that was started earlier but which was never a success story—Cetus—C-E-T-U-S—over in Berkeley and then going to Genentech. And Genentech was mean and lean. Cetus had—they brought in a guy named—well, I forget his name for sure—but they brought in the chairman of the board. It was a very fancy lunch. And Genentech was a bunch of guys in tennis shoes trying to clone genes. I remember coming out of Genentech, and Mary says to me, after we had been to both the same day, "So which one are you impressed with Jordan?" And I said, "Cetus. That is really an impressive place." She shook her head. She was a lady of few words. She shook her head, kind of frowned, and said, "These boys got it." That was Mary Lasker. "These boys got it." Those were her words, and she was right. Genentech became a multi-multi-multi-billion dollar company, which is now owned by Hoffmann-La Roche. It is still a research subsidiary. And Cetus is nowhere to be found. Mary got it. You know, she could just see this stuff. It was amazing.

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So then I got the hook of biotech. Again, frankly, at MD Anderson in the early '80s, nobody was cloning genes. It wasn't—you just—it wasn't a thing you did in academics. I could see what biotech was doing, and of course the big pharma, and I recognized that some of the pharmaceutical companies—not all, though—they were very slow getting into proteins and cloning. Roche was early, and Schering-Plough was early.

Tacey Ann Rosolowski, PhD

0:43:26.9

Why was academic research so slow to pay attention?

Jordan Gutterman, MD

0:43:34.1

I'm not sure. I'm not sure. Although—well, I'm partially inaccurate here because Mike Bishop and Harold Varmus, who shared the Lasker prize in '82 and the Nobel Prize, I think, in '89 for cloning oncogenes—discovering and cloning oncogenes—were doing cloning. But they were in San Francisco, and that is where all of this was going on at Genentech, at least—you know—in the Bay Area. So they were doing cloning. Let's put it this way—a little more accurately—MD Anderson was not doing it, because I remember coming back from the Lasker awards in '82, I think it was, when Bishop and Varmus and Ray Erikson, Bob Gallo, and one other person—I don't know—I think it was Dale Kaiser—shared the Lasker award. But the key ones with oncogenes were Bishop and Varmus who won the Nobel Prize. They were in San Francisco. I remember coming back and talking to some colleagues of mine saying, "We have nobody working on oncogenes here." I mean, we got—these are cancer genes. And there was one guy who stayed another few years and then opened a B&B in the state of Washington. I haven't seen him since. Now, of course, that has all changed, but we were rather slow in basic research. We were rather slow.

Tacey Ann Rosolowski, PhD

0:44:45.8

I am sorry. That was a funny little move there. I am just going to open a B&B. (laughter)

Jordan Gutterman, MD

0:44:55.1

He couldn't get grants. And I was frustrated with that. So I looked wherever—and the same with my new one too.

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Tacey Ann Rosolowski, PhD

0:45:03.7

Now, let me just ask you, was he not getting grants because he was working on oncogenes and nobody was seeing it, or—?

Jordan Gutterman, MD

0:45:09.7

No. Well, I'm just not sure he was extremely good. He was smart, and I liked talking to him, but he probably—and I don't know why, but I think he just couldn't get the support he needed. But we were way behind the eight ball. But—you know—here—but I am not criticizing because this place is powerful clinically. We've never developed a hugely competitive basic science. There are some very good basic scientists. But I will go anyplace. I don't just stay within these walls. I never have. I will go anyplace in the world, or companies, whatever it takes to put the pieces together that I have to figure out we need, because I like collaborations. Not everybody is that way, but I do—I'm not threatened by it, and there are a lot of people a lot smarter than I am—but putting the pieces together.

So I could see that big pharma with especially Hoffmann-La Roche had this amazing thing of purifying proteins and then trying to clone the genes. Don't forget, in the '70s this was not done routinely. It was certainly not commercial. As I said, the first commercial product of recombinant DNA was insulin, and it was put in the clinic, I think, just a few months before interferon. That was a big deal. That is why Time did that cover story at the end of—March 31, 1980—"The Big IF," but it was—and that is a very, very balanced article. When you see it on the cover of Time, it gets played—you know—oh my God—all this hype. But people, if they are intelligent, are going to read this and try to understand parts of it, you know?

Tacey Ann Rosolowski, PhD

0:46:42.7

Uh-hunh (affirmative).

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Jordan Gutterman, MD

0:46:43.0

And people—you can't—I mean—and we need to communicate more. I think that has changed, by the way. I want to stay on track here, but you asked me—I think that has changed. Scientists are much more—completely different today. They are more willing to get their names in the paper and explain the science much, much, much more. It is much less frowned upon, in my opinion. Now, if said incorrectly with hype and blah, blah, blah, of course. So I think that's a really good thing because the scientific education is tough. It is tough for us to understand stuff because science is moving so rapidly. Technology is moving this still so rapidly. But I could see then back—

Tacey Ann Rosolowski, PhD

0:47:19.2

Let me just clarify for the recording, because you were following up on a conversation that we had before the recorder was turned on, we were talking earlier about how a couple of decades ago it was a different attitude about doctors who would actually speak in the media. It was actually frowned on with suspicion.

Jordan Gutterman, MD

0:47:33.1

Yeah. Well, doctors being people who see patients as opposed to basic science, there is a little bit of difference there, but go ahead.

Tacey Ann Rosolowski, PhD

0:47:43.5

No, I just wanted to clarify things.

Jordan Gutterman, MD

0:47:44:3

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Yeah. Okay, yeah. Good. Okay, I understand. So whenever I went to Genentech I just—I mean—I was so excited to walk in those walls and see the power of venture money. And yes, I'm sure as they got bigger and bigger there was more bureaucracy, but I can tell you Bob Swanson and Genentech—they let those guys work. And that's me—you know—freedom with as few restraints. Obviously you have to have some boundaries, less so at Roche. But again—and in part it was because they had the resources and they were under a certain amount of pressure to produce because they had the money. I loved that. I loved the ability to accomplish—the freedom. That is why I respond to art a lot, because these guys I always used to envy—I still do—Sam Francis, Mark Rothko, Richard Diebenkorn—because they can go in the studio if they have enough money to buy paint. There is no committee. There is no oversight. You can do what you want. If you're a writer, you can write. Now, you do have to get the approval of people so you can maintain that, right? You have to sell your poems. You have to sell your literature. You have to see your essays or your art or your music and so forth. But I always envied the creative—the artists. It's that part of me—that side of me. And I think that's true for a lot of scientists—Joe Goldstein—a lot of Nobel Laureates—Mike Bishop—most of the Nobel Laureates I know that I work very closely with and have become friends with, with the Lasker award—Lasker jury.

One of Mary's greatest legacies, if not her best, was getting me involved with that. Because I can work with these people, and I see how they think, and I see how they live and so forth. It's amazing. Most of them are creative people who love the arts. I think that can be so in science if you can construct your environment to have creative people in however you put the pieces together—being very careful with conflicts and paying attention to the I/thou principle that you are not hurting people and so forth. You don't need to. But—you know—the bigger and the wealthier institutes give the more rules—the more regulations. This comes in part through growing up in a household of a father for which the government and the rules say, “You're Jewish, and you're not going to have access to this. And you can't go to school. And you serve in the army or we'll kill you or we'll rape your mother,” or whatever they did. I have a difficult time with too many constraints on my creativity and ability to do things because I think I am doing something that really counts or will count. And that passion—it's more than passion. It's intense. I think this is shared by a lot of people, this type of thing. So I was so excited—I still am—about going to any place, whether it is MIT or Harvard or—but it could be Genentech or a pharmaceutical company that has the power. Anybody intelligent doing creative stuff just—it is just the biggest excitement and so forth. And again, biotech and pharmaceuticals at that time—now things have changed, and we will talk about that a little bit later.

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So that is how I first started getting involved with—there was no way to get recombinant interferon. We couldn't keep buying stuff. Eventually we had enough where everybody then could do the hairy cell. So in '86 the drug was approved. It was rather anti-climactic, and I wasn't even sure what would happen next. I never had this experience. Then in the fall of that year, I get a visit by a woman who actually taught me a lot. Her name is Meredith Grimm—she may have changed her name by now—an attractive woman from Schering-Plough. She came to my office, and she said that they wanted to do some post-approval marketing and so forth with interferon. I really didn't probably understand why she was here. What she was here for was once it gets improved, you want to start enhancing the indications—the use of it. She was very creative about this. What she says is, "We can offer a small grant to a clinical investigator—enough to pay, say, for a nurse—that would do a study in kidney cancer with our drug or in these diseases that have not been approved to get enough data to try to enhance the label because of reimbursement. Because although you can get reimbursement without being on the label, that is called off-label use." So starting in '86, and going for the next several years, I got very involved and very intrigued by the whole business part of making drugs.

I work with a guy named Steve Huber here, a research pharmacist—I think he is a PharmD—about reimbursements. We have fascinating problems. I'm not going to talk about them right now, but we worked out a lot of issues, even issues regarding patients who would come here who could not afford the interferon for an indication that was really needed, including hairy cell and others. Reimbursements where the company would have a certain supply to give to MD Anderson in exchange for things, that's so patients could have access, so accessibility to this expensive drug. Don't forget that after insulin it was the first recombinant protein for us, approved by the FDA. There was an antibody—okay—I forget the name of it now. I think Ortho Biotech made it. It was produced in part by recombinant DNA, but interferon was the first recombinant—really the first—the second recombinant molecule, I think, approved by the FDA. So this was all new ground, and it was interesting. It was fascinating.

Also, we had enough drug, by these little grants, to begin to expand use. And Schering, in part because of Meredith and I'm sure other scientists there—she was a nurse—is a nurse. I haven't seen her in years. She left Schering. But because of the people at Schering—that I began to work now—see, I had worked with Roche before. Now I began to work with Schering in this issue and consulted with them. They decided to go after hepatitis C because a guy named Tom Merigan, who co-did the first recombinant study with me from Stanford—a virologist—had shown previously in a few people in Europe—individual patients with hepatitis—that crude interferon could work. Roche, for some reason, made the decision to not go after the market. That is where the big money came and the big use, because if you asked me today what is the biggest impact of interferon in patients, it would be hepatitis C.

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Now, there are more drugs—Gilead is a major company that has produced drugs. But interferon, intron, other interferons are still a mainstay. The trouble is toxicity, but it's still the natural defense against viruses, so interferon is not going away. It's just—like a lot of drugs, it's going to be other drugs in combination or supplanted and so forth. Now, eventually Roche got into it. So that was interesting.

And I remember—fast-forwarding a little bit—I think it was '82. I was in Chicago presenting, and I—well, around that time in the—not in '82, '92, but in the late '80s, I remember the first person to call me was from Kidder Peabody. I didn't know what Kidder Peabody was. It was a brokerage company, I guess. They were having a meeting, and they wanted certain academic people and company people talking about molecules—growth hormone from Genentech, EPO—the erythropoietin and the red cell thing, and other drugs—interferon of course—and then others as they came along. That was my first exposure to one of these meetings where all these biotech people and analysts trying to predict stocks were in the room, and that was fascinating. They would listen to every word you said. They would question you. Again, it was just a whole new world of analysts—mainly analysts—trying to predict which drug company, which biotech company has got the use—what is the market going to be for interferon and so forth. So I started doing some of that. It was very fascinating, very interesting.

Tacey Ann Rosolowski, PhD

0:56:23.6

That's another system.

Jordan Gutterman, MD

0:56:24.6

Yeah, exactly. Then I remembered going to Chicago with one of these meetings, and I presented. A guy stands up, and he presented a curve. I can't remember who he was, but it was in Chicago. I'm sure I have it in my notes—my oral history. And he shows how interferon in 1986 made \$30 million apiece for Schering and Roche, something like that. Because that was always the thing—well, this is no big deal. I mean, The Wall Street Journal didn't really care about it. Hair cell leukemia is a small market. They didn't recognize there would be off-label use. There wasn't a lot of competition, by the way. Today there are so many interesting compounds for cancer, but then there was very little else to do but chemotherapy. So there was a lot of off-label use, and newer indications—the way Schering was approaching it—were beginning to emerge. So in '92—six years later—they showed the first year. And even though I had been to these analysts meetings, I hadn't really paid much attention. It never was on my radar screen. The first year was \$30 million, and then it was \$60 million. And then in '92, if my memory serves me correctly, he showed it had reached \$1 billion. I think that's US, but it could've been worldwide—I'm not sure—a combination of Hoffmann-La Roche and Schering-Plough. Man, was I excited. I remember several people saying, "You must be a rich man." I said, "I haven't made a penny out of this." It just—

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Tacey Ann Rosolowski, PhD

0:58:03.6

Why did the money excite you? I mean, why did seeing that increase excite you so much?

Jordan Gutterman, MD

0:58:07.7

Because, first of all, I think there is a piece of a businessman in me. And of course, it wasn't my money, but I think the most important is if you can make money for a company—a pharmaceutical company or a biotech—they are going to do this again and again and again, because if it didn't make money there would be no incentive. I mean, it's just the way it is. And that is a whole dichotomy between the business world and science and philanthropy, which we may not have time to talk about because I did this largely with philanthropic money. I didn't make any more. I did it with Mary Lasker, the Interferon Foundation and this Clayton Foundation, which we haven't talked about as well which is extremely important.

And the whole foundation scene in Houston—you asked about—we will probably not get into it today—what is it about Texas and MD Anderson and so forth? A lot of it are these wealthy foundations, many of them started by bachelors who had no relatives other than nephews and nieces who had made all of this wildcat money to oil money. I remember recently this man who came to see me thirty years after his wife died—thirty-three years. He was here in Houston with his new wife, looking around, looking at these buildings—I walked him around—and he just looked and said, “The beauty of free enterprise,” because he sees the money. This was not built on academic grants. This was built by philanthropy, these buildings. Of course, there are a lot of business aspects to it, but money—

Mary Lasker said something—I still—there are three things that I still remember that she said that just are stunning. One of my favorites is that, “Money is frozen energy.” So that is why it excited me. I could see money as frozen energy; it just releases all this stuff. Another thing she said which is—what she did when she took me to Roche and Genentech, and then it was on—you know—if I was not very effective, it would have died right there, but she could see I was effective even though I am very shy, I think, personally. But when I talk science, or I can talk—I can get going once I am comfortable. I know those roots—I know why I am that way, where I grew up—where at first, when I meet someone, I am extremely—just shy. That is the little boy in me. That is my analytic thing.

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But her—another one is, “The greatest gift you can give a person is another person.” I mean, that is profound. It is so profound. So she gave me all this stuff. It’s still ongoing. It’s like life. It’s like my father, my father’s ethics and his history, my mother, and a lot of other people—Rounds with Mom—they’re still alive. I mean, Faulkner once said that, “The past is not dead. In fact, it is not even the past.” You’ve got to think about that. “The past is not dead; it is not even the past.” I think that is why when you live, if you can make the most of affecting people including frozen energy, so—and in a way that could be the title of a book—Money is Frozen Energy—because Texas has all this frozen—I mean—all this oil. All this money came in, and what happened is this—Texas Medical Center. I didn’t understand this, of course, but it was intuitive to come here maybe. There is MD Anderson, meeting Mary Lasker. All this stuff is just constantly being extracted without fracking out of the ground—all this energy. It involves money, it involves science, it involves brains, it involves social stuff, and it involves philanthropy. It’s just amazing stuff. Okay.

Tacey Ann Rosolowski, PhD

1:02:02.3

Was there any downside of working with a drug company?

Jordan Gutterman, MD

1:02:06.4

Oh, yeah. Oh, God—okay. So let’s talk about that. You want to just click it for a second?

Tacey Ann Rosolowski, PhD

1:02:12.1

Sure, no problem.

Jordan Gutterman, MD

1:02:12.6

I want to just take a deep breath and look at my notes.

[The recorder is paused.]

Tacey Ann Rosolowski, PhD

1:02:15.7

So we are back after about a five to ten minute break. It is 2:37.

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Jordan Gutterman, MD

1:02:21.8

Okay, you asked me about the downside—the potential downside of working with the—with industry. First of all, for this stage in my career, even though I had a small laboratory and in ‘86 became a chairman of the department—a large department with a lot of different clinical scientists and laboratory scientists—I still was primarily a clinical scientist. And companies make drugs. I mean, it’s very difficult for an academic institute to make drugs, and they should make drugs. So on the one hand it’s very, very exciting, and they have the experience of making drugs. I mean, people don’t understand the complexities, and that’s why, in part, the cost is so high. But to make a drug—I just love reading the history of drugs. And I’ll get to your question about the downside.

But I remember as a kid—again, back in South Dakota—everything always dates back to the same thing. Like a lot of people say everything can be pointed back to Seinfeld, I think everything can be pointed back to this little town in South Dakota. And for me, I’m just saying the reason I always go back is because I have a hard time with patience or people when I don’t know what they’re all about. I just don’t fully—I have to get down to the depths of it. I really can’t—I have a hard time when I meet new people. I know I’m digressing slightly. But when I meet new people, and I think I’m going to work with them—whether it’s a company guy, person, whomever—I always want to start out where are you from and get to know them a little bit. I can’t work in a vacuum, but a lot of people aren’t that way. They just want to get down to business. I don’t do well with people like that. I just can’t work with people like that. There has got to be some human connection. There has got to be sort of an I/thou relationship—you know—as opposed to I/it.

So my dad had this store in Flandreau—and this, by the way, characterizes a lot of what you’re going to have when you ask me a question. It takes me a while to get there, but I will get there. I’m not thinking about it. I’m just spontaneously free-associating. But the thing is, next door to his was a pharmacist. And the wife of the pharmacist was the daughter of a doctor that my parent’s house—he bought the house. Anyway, they were very close to the pharmacist—Mr. Roth and his wife. My brother and I—my twin brother and I—we would buy our little comic books, which I still wish I had today, of course, like everybody—Captain Marvel, Batman, and all that stuff. But I can make my interferon money, right? But anyway—and we are going for a cherry Coke and stuff like that. But I remember my dad, in high school, saying, “You know, I think you’re going to become a doctor. I think you should go to pharmacy school first. I think you need to learn how drugs are dispensed and made.”

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Now, my father had zero background in medicine or science. He was an immigrant. He worked—you know—he owned a grocery store and a department store. That is what his talent was. How prophetic, and did that influence me to make drugs? Because this new one—we have actually discovered a drug. I mean, we have actually started from the discovery, and we're all the way, ready to go to patients. We'll tell that story another time. I mean, this is really a story now because I found this stuff. We found—we discovered it out of a plant. Is it because I want to make my dad happy? I don't know. I'm just sitting here analyzing it because he wanted me to go to pharmacy school. I mean, I still don't understand that. But I did have this sense of this pharmacist dispensing drugs and grinding them up the old-fashioned way and stuff. And I would see this. I hadn't actually thought about that too much until just this moment, but the influence of Mr. Roth and Lillian, his wife, and someone who eventually really did do drugs. But part of it also is because, I think, my nature is to understand, but understanding for me without helping—without translating it—the physician side of me just probably can't deal with that. So to understand something for the sake of the understanding is wonderful, and it is very exciting. I love it. But it's not complete. It's not enough. I feel almost guilty, like what are you going to do with that?

Now, a lot of basic scientists won't agree with you, and they shouldn't agree with me because a lot of the—some of the most amazing discoveries in science have been with no thought of application. This is a constant theme in science, and I agree with that. Scientists should be free to create like an artist. You never know what—you know—without the thought it could be useful, you know? And that is the same with inventions and so forth. But as a doctor or as a person, for me the greatest joy or the incentive is to—can I take my brain and my understanding and my learning and one day maybe help somebody with it? That's just me. It doesn't make it—I'm not better or worse than someone who doesn't think about application. And I don't always think about it.

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But I was thinking about that pharmacist—Mr. Roth. I have amazing memories since—you know—very emotional about him in his little white coat and grinding with the mortar and pestle, and my mother going in. At the time, when I was a kid in the '40s, antibiotics were just being made, you know? Penicillin and sulfa, as a kid—you know—I had those drugs. I was sick, my mother would call, and my father would call Mr. Roth, or they'd just walk next door and get the syrup or whatever, and I'd feel better—for a cough, or if it was ipecac—you know—the old drugs. Then I would see these people on rounds with mom, and sometimes medicines would help, and sometimes they wouldn't help. So I think it's natural for me to have realized and to have gravitated in part because of Mary Lasker introducing me—that without the people that make the drugs—that is the industry that makes the drugs. I mean, Boeing makes airplanes. Schering-Plough doesn't make airplanes. Boeing shouldn't make drugs. You need that industry. We have to protect industry. It's a great American triumph, is that industry. And we could talk about what is happening now. It's changing, but things change. And then biotech came out of cloning genes, which was there with interferon starting in '73. The power of being able to take a gene, make a drug out of it, and see a patient get a platelet count and live—I mean—that is amazing. It's just amazing. That's the positive side. Now, let's talk about the downside of the question. See, I get to the question.

Tacey Ann Rosolowski, PhD

1:09:40.8

I always knew you would.

Jordan Gutterman, MD

1:09:44.2

At first, with Roche—when I went there in June of '78—the first three years when they were trying to clone and so forth, it was great. It was a challenge—it was—every time I went. But now when you deal on the clinical side, other elements come in, although things have changed a great deal. But when I first started doing this, many but not all—not the ones I mentioned, actually, but some of the physicians are pretty programmed. I mean, that's what they have to be. They follow rules. They follow FDA. They are very conservative, very cautious. Not necessarily some of the ones I have mentioned who were the leaders, but down the ranks—but not all. There is a mix. Whereas I am curious and want to be not reckless, but take risks and understand things, they are very deliberate, and they have to fill in boxes, basically. And they have to be careful with the FDA and so forth. They are more conservative. I think it's the conservative nature of the clinical people that really bugs me. Not all of them. Genentech, again, had—we became great friends, and we're still friends today with many of them that I work with. And I still am friends with many of them. They are wonderful, wonderful people. But some are too conservative. Again, it's part of the job. I mean, they have to—

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Tacey Ann Rosolowski, PhD

1:11:05.5

And how do you define conservative? Give me an example.

Jordan Gutterman, MD

1:11:08.6

Cautious. I like to do hairy cell leukemia with a recombinant. Well, it's a small market. In fact, that was the downfall of a lot of companies. I've consulted with Biotech. There was a company in Colorado—Synergen—that went after the wrong target because they went after the biggest market when in fact they could have done with interferon and gone after a market that would have worked, and then after getting it approved, go after the bigger markets. That still has not caught on. I wrote a paper on the nature of biotech in 1987. I think it's been referred to once in the literature of how that strategy backfires. If you only go for the big markets—and now with personalized medicine, these markets are going to be smaller anyway because these—we're not going to have these massive trials. We are going to have to focus. So that is one of the downsides, I think, of a conservative nature.

I don't do well with rules and bureaucracies and the time it takes. Now our institute has become horrendously—one of the reasons I gave up a department and went to the lab is—and I'll tell you, we're not going to talk about it today because I thought there was a new way of going, because of my interest in nutrition and plants. But it also was getting very difficult to get protocols through and the layers and layers and layers of review, in part because of the lawyers. Everybody is afraid of liability and so forth and so on, and that is very strenuous, very, very taxing—very taxing. So it's not just the institute, not just the industry. I think it's a conservative nature.

I mean, if you wanted to go after hairy cell, they're not going to do it because it's not a big enough market, even though it may not be the right strategy. But with interferon, I worked first with Roche, and then Schering. After it got marketed, we had a lot more freedom because the drug was out there, so we could do more of what we wanted to do. I find that Biotech was more risky. I mean more—excuse me—bolder because they had to be. They were younger, leaner, so to speak, and much less established in the years of bureaucracy. So that would be one thing.

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And we worked with several companies—this GM-CSF with Immunex and Schering-Plough. That went okay. It went okay, but I wasn't terribly excited about the work. We also worked with Genentech on a couple of compounds that went—did not go anywhere—gamma interferon, another interferon which is really a misnomer. It's not really anything like interferon-alpha. And then TNF—tumor necrosis factor—these turned out to be what—especially TNF and then interleukins—especially TNF turns out to be a cause of the disease of rheumatoid arthritis. It doesn't really help anybody. It makes people sick. So a lot of this—as I said yesterday, many of the cytokines turned out to be causative of diseases, although if you count these growth factors as cytokines, they do help people's blood counts. So it's a mixed bag.

There is a whole field out there of Immunex and other companies—Synacor and so forth, Amgen—who now try to block these cytokines for disease, and they are highly successful. Enbrel is an example of an Immunex—now it's an Amgen—product that blocks TNF for rheumatoid arthritis. Working with them was much easier, I must say. Working with Biotech was much easier. It's just personality, you know? My personality is discovery and so forth. They have to follow certain boundaries to get drugs approved. It's just the nature of the game. The nature of someone who would work in a pharmaceutical company and just carry out somewhat these tedious things of writing protocols, getting FDA approval, following all the rules, setting up all the studies, it's just—that's not what I do. So it's just a different personality. So that is kind of the downside, maybe.

That's why I worked—that's why it kind of explains, maybe—more than maybe—why I told you I did two parallel tags. Fortunately we had enough drug left over we had raised enough money with the Interferon Foundation—not expecting and not knowing what it would be cloned or synthesized—so we actually raised more money probably than we needed, and we had more interferon because—in fact, I was concerned in the '80s—oh my God—we're not going to be able to raise any more money. Everybody is going to say, "The companies are making it." And you know what I said many times? I said it to Mr. Davis—Leon. I said it to Elaine. I said it to others. "Don't count on them to make more discoveries. They don't always make discoveries. Now, in targeted therapy, if they know ahead of time, that's one thing. But if you want to make discoveries, don't count on the companies to make it. We need to have our own interferon." And I was right. That maybe characterized as the downside. "Don't count on it," I said. They're going to do what they have to do to get this drug approved.

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We found—listen—we found the hairy cell leukemia. Very few people understand or appreciate that history. Everybody thinks Schering and Roche did it. We found it. I found it. My colleagues at MD Anderson found it with private money from Texas and oil companies. That's how we got it. So Schering and Roche, overall, made billions on this drug. And it was discovered—I mean—the activity was all done by private money in the state of Texas, period. So we need them, but don't completely rely on them. And that is the parallel track, which at some point I figured out. "Wait a minute; they're going to drive me nuts." It's so boring. You get up in the morning, well, what does Seymour want me to do today from Roche? That's the negative—as opposed to being able to paint a picture. I want to paint a picture. I don't want to do it by the numbers. I want to paint a picture. I hadn't thought about that. That's a pretty good one, actually. I don't want to paint by the numbers; I want to make my own picture. Okay? Does that answer the question?

Tacey Ann Rosolowski, PhD

1:17:24.8

Yeah, that's a good. That's a good answer, a revealing answer.

Jordan Gutterman, MD

1:17:30.4

And I guarantee you we're doing it now, because we have our own drug, and that's a whole other set of things that we'll talk about some other time. But when we start talking about venture capitalists and starting a company and giving up the reigns, losing control, and how you do all this stuff, that's going to be an interesting story. But we're not going to talk about it today. So that's kind of the downside. I want to paint my own picture. I don't want to do it by the numbers only. I will do some. So why don't you ask me some questions?

Chapter 10

B: Key MD Anderson Figures

R. Lee Clark, Charles LeMaistre, and Philanthropic Houston Oilmen

Story Codes

A: The Researcher

C: Portraits

D: On Philanthropy and Volunteerism

D: On Research and Researchers

D: Understanding Cancer, the History of Science, Cancer Research

D: The History of Health Care, Patient Care

C: Professional Practice

C: The Professional at Work

A: Character, Values, Beliefs, Talents

A: Personal Background

B: Critical Perspectives on MD Anderson

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B: MD Anderson History

A: Experiences re: Gender, Race, Ethnicity

C: Critical Perspectives

A: Obstacles, Challenges

C: Discovery and Success

Tacey Ann Rosolowski, PhD

1:18:12.9

Are we at the point where—in terms of you talking about your research—where we can talk about you moving away into the area of Avicins, or are there more pieces to put together?

Jordan Gutterman, MD

1:18:24.8

There may be, but maybe it would be after I have a chance—it's going to be a lot of stuff to read.

Tacey Ann Rosolowski, PhD

1:18:30.0

Okay.

Jordan Gutterman, MD

1:18:30.4

At some point maybe tap into my own oral history where I will fill in blanks. I know I will. I've left out probably some things that may be important both in terms of MD Anderson, and I am quite aware that this is an MD Anderson history. It's not just about me, but how I did it within the confines. I think one of the things that I have a gap in right now, and I probably will introduce this topic without getting into it, is what have I missed about MD Anderson? I think a lot. I think I need to talk more about that, but it may not be today.

Tacey Ann Rosolowski, PhD

1:19:04.1

Okay.

Jordan Gutterman, MD

1:19:04.5

I'll think about that.

Tacey Ann Rosolowski, PhD

1:19:04.8

Okay.

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Jordan Gutterman, MD

1:19:05.6

How all this stuff I'm talking about—as I reflect on what I've talked about—meanwhile, I'm here. I'm a professor, and I'm doing stuff. So how does this relate to the political, sociological environment and so forth and so on? The changes from one president to another president—from one department to another department—

Tacey Ann Rosolowski, PhD

1:19:25.7

Well, actually, you are being very prescient, because I just pulled out that whole roster of presidents that you've worked under, and you've worked under four of them now.

Jordan Gutterman, MD

1:19:37.3

Four now.

Tacey Ann Rosolowski, PhD

1:19:37.8

Yeah.

Jordan Gutterman, MD

1:19:39.3

Three and half, but that is beside the—well, DePinho just started.

Tacey Ann Rosolowski, PhD

1:19:42.6

Yeah. So I was wondering if you could kind of describe the marks they each left on the institution—you know—maybe if you had personal interactions with you dealing with an issue, how that worked out, your impression of their working styles. And every lead has both strengths and weaknesses, so what did they bring, really, to the institution?

Jordan Gutterman, MD

1:20:07.1

Well, I'll talk about Clark. I'll say something—some things about Dr. [Charles] LeMaistre, not necessarily particularly favorable, in terms of my interaction with him. There was one thing that I probably will describe, but I'll have a chance to see this before—?

Tacey Ann Rosolowski, PhD

1:20:35.0

Yes, absolutely.

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Jordan Gutterman, MD

1:20:38.9

Dr. [John] Mendelsohn came at a different time. And some of this may be better when I describe what I—because Mendelsohn came exactly the same year that I changed into a laboratory person and kind of went underground. I'm serious now. I've gone underground for, gosh, sixteen years.

Tacey Ann Rosolowski, PhD

1:21:04.7

Wow.

Jordan Gutterman, MD

1:21:07.3

I haven't talked to anybody about it much. We've published a lot of papers. So during the whole Mendelsohn era—he came in '96—that's when I started with the Avicins—we discovered the Avicins. Mendelsohn left in 2011—you know—2012, and so that would be much less. And I kind of—I went underground. I just became—I became a recluse. People ask me, "Well, why did you give up all that blah, blah, blah? Everybody knew you. Why would you take a chance and start something brand new?" It was completely abstract. The Clayton Foundation deserves a lot of credit. We will talk about that.

So Mendelsohn—there won't be a lot, but we will talk a little bit about that because he and I crisscrossed way back in the interferon days. DePinho, it's too early. So I will talk a little bit about Clark and LeMaistre and also this transition. First Clark, because it does deal with interferon, and then LeMaistre was interferon. Then Mendelsohn came, ironically, as I think about it, exactly when I changed fields.

Clark—well, I was very young when I came here, but I always found him to be just a wonderful, wonderful gentleman. He was just a gracious, gracious man. I came here in '71, and in '78 when all this burst open, he was on the original cancer panel when— Mary Lasker, in '71—it turns out, the year I came here, Mary Lasker pushed Nixon's administration through a guy named Bobst— Elmer Bobst—to pass his national cancer plan. She was convinced that if we put in enough resources we can—and even though people have highly criticized the nature of it, because she thought we could have cancer cured in five years, ten years, twenty years. It's much too complex, if we can ever really cure the disease. Cancer is always going to be with us, every time a cell divides. But what she did, I think, was quite visionary.

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She worked with Clark quite a bit because Clark, at that time—see, don't forget now, this is the part about Anderson—and we may not finish all this today—is to go back in time when I'm doing all this stuff. What was the nature of society and MD Anderson cancer research? So in '71, the year I came here, was the time the national cancer plan started. And again, a lot of this was because of—I could see how well trained Victorio Rodriguez was in clinical cancer, and that was what I wanted to do. Again, I'm not sure why exactly. I thought about this last night.

[redacted]

I became a hematologist. I wanted to do blood diseases, but he introduced me to solid tumors more. And in fact, there was no profession—no specialty called oncology when I first came here. It was just started the year I came. So a lot happened the year I came here. But I knew this was a place—I could see this was a place I could do stuff. I'm not dreaming about any of this stuff.

When I came here, I thought MD Anderson was already massive. I thought Houston was massive. And—but I met him pretty early, and he was such a gracious man. He was originally, I think, from Georgia, but—you know—he embraced the Texas culture. He had a ranch, and it was a small place. Almost everybody knew everybody else. I mean, our HR department was just down in the main campus. You would just go down there—if you wanted an employee, you just go down there and talk to Ruth and say, “You know, I need someone who is this and this and this,” and they would find the person for you. And that is—because he retired in '78. It's funny; these presidents have come in—I'm forgetting DePinho, which I would like to do. Jesus, is that—that goes nowhere.

I came here in '71, the interferon broke in '78, and that was the year Clark retired. I switched fields in '96, the year LeMaistre retired and Mendelsohn came in. And I'm starting a company here in Houston in 2012 when DePinho comes, which keeps me somewhat independent of this place. So I think—I always have kind of a—maybe it's, again, like my father. When you're in that situation with the czars and all this type of thing, you've got to always be ready to go. It could be my background of being Jewish—always have your suitcase packed. When is the time you push your sons out to go to America because I don't know if we are going to make it? I wonder. I'm not—I mean—I've been here forty years, and I'm going to be here another—whatever. But you have to have backup plans in life, I think. I don't know if I'm making sense here, but strategies.

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So with Clark I never felt that. I was young. But what I really loved about him personally was in—he knew Mary Lasker. Mary Lasker would praise me to him, and she had dealt with him with the National Cancer Plan and became one of the three advisors to President Nixon on the National Cancer Plan. There are wonderful pictures around here. So he and Mary were very tight. She was very close to people like this. So I was—when she met me in '73, '74, I was kind of someone he knew and supported immensely. And then in '78, as he was leaving—before he left—he really was excited about this—about the interferon and Mary Lasker's involvement. It was quite a story even then, before we had done too much. But he went to Cuba. He got Castro to start making—in the seventies, everybody was trying to make interferon because nobody believed it could be synthetically made—that is, by cloning. So there were companies all over the place with blood banks trying to make all this precious stuff. I'm not sure I've talked about that, but we won't talk about it today. I could review my notes on that. It's been a long time. But Clark was very involved with Cuba.

Tacey Ann Rosolowski, PhD

1:27:19.2

So what was he doing there?

Jordan Gutterman, MD

1:27:20.8

Well, to get Castro—he had some relationship with Castro, but I think he was trying to get Cuba to start making interferon, maybe to commercialize it and stuff. I don't know. I have to go back to his history to find out. But I remember the year that he retired, in '78—Mary Lasker and Ann Landers—Eppie Lederer—came down to visit DeBakey and Clark as he was leaving. I gave a little talk up in the conference room that still exists upstairs, over the main campus. Mary was there, Eppie was there, Clark came, and LeMaistre came. LeMaistre was just coming in and was introduced to this whole thing.

I love Clark. I mean, I just—and then it was interesting. I was living in the high-rise after he—many, many years later—he had already retired, of course, for many years.

[redacted]

I had a very, very warm, very fun relationship with him. He was a real person of—he cared about people, and he was a visionary, just an amazing visionary. Those were much simpler times, though.

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Now, LeMaistre—we may say more about Clark, but LeMaistre, I think he was concerned about all the publicity. I think he heard bad things about me. He was a different man from Clark. I think he—I can't—I don't know this for a fact, but I think he was concerned whether we were too far over the top. I mean, there was the press here all the time. These were heavy days with cloning. NBC and CBS and—you know—all this stuff was happening all the time—local news and papers. It was hard to avoid that. And then we were getting these results—tumors were shrinking, hairy cell leukemia, CML. We were reporting this stuff, and people were picking up on it. I mean, it was real, and it is real.

Tacey Ann Rosolowski, PhD

1:29:31.9

Were there ways in which he overtly demonstrated support or concern about that?

Jordan Gutterman, MD

1:29:36.9

Yeah. Well, what happened was—now, this is where women are interesting. When I met with Elaine Davis—and we've got about ten to fifteen minutes. We will probably just get to this. We're not going to get to the transition today.

Tacey Ann Rosolowski, PhD

1:29:47.8

That's fine. Next time.

[redacted]

Tacey Ann Rosolowski, PhD

1:31:02.5

I'll just pause this.

Jordan Gutterman, MD

1:31:05.4

Okay. Sorry. I should have shut that.

Tacey Ann Rosolowski, PhD

1:31:06.4

That's okay.

Jordan Gutterman, MD

1:31:07.2

Ready?

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Tacey Ann Rosolowski, PhD

1:31:07.5

Yes, we're ready. We're good.

Jordan Gutterman, MD

1:31:11.0

"I don't want this place getting a penny of it." In fact, when Mary gave a million dollars to the Finnish Red Cross to buy interferon, she just sent the money directly to Finland. But she sent it in '78, just as LeMaistre was getting here, and that doesn't go well with administrators. So we got a million dollars, which economically was an amazing thing because we were getting people—in '78, when the publicity came—we were getting more people than we could possibly handle. And we weren't charging for the drugs, and they were paying to be patients here. But I'm guessing now that administrators in general, not all, don't like that loss of control, because the money never went through here. She said, "I'm not sending it there. If I send a million out there, I might get a half million back out. They'll have some rule or make up some rule and take it." This was Mary. Elaine Davis, not knowing that, said the same thing. Women—intuition. It wasn't a trust factor; it was just knowing how the world works. And she said, "We're going to have not one penny go for anybody. We'll pay for a secretary, whatever it takes, but we're going to buy material for you—frozen energies—so you can treat patients." They didn't care about the research aspects. They just wanted to treat patients. They were rather naïve about things, but—you know—they are not scientists. So that is how they started the Interferon Foundation.

And they brought in a guy named Roy Huffington from Huffco Oil—a billionaire oilman— independent oil dealer who had mostly his oil in Indonesia. That was my first exposure to the oil people. I went over to see Roy Huffington and his wife. He gave recently—before he died at the age of ninety—two or three years ago—he gave a big amount of money for an aging institute over at Baylor—Roy Huffington.

[redacted]

He was the—Leon did kind of the legwork on it. He was the soul behind it among the men in this foundation. But the entrée was Roy Huffington. He knew everybody. And he was a Texan. He had all the entrée to everybody.

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We started then going first to Shell Oil. We met with John Bookout, who was then the CEO of Shell. Leon and, again, Elaine were so smart about it. They said, “We’re not going to bureaucrats.” You know, it is kind of what I said about the downside of big pharma. “We’re not going to the head of the foundation. We’re going to the CEO. We don’t want to have to work through all that. We don’t have time. People are dying.” And this was in ’79, ’80. The foundation, I think, was incorporated in early 1980, which, by the way, was exactly the same year that interferon was cloned—not producing the full-length drug—and I’m thinking we’re screwed. We’re not going to raise anything with all this PR going on at the same time with interferon. But we did. We got it done before it became a drug. It took a year, but in that year we raised all the money we needed to eventually translate it into all these saved lives. I didn’t know all that. So the timing of everything was amazing.

So we went to John Bookout. Now, I had been on McNeil/Lehrer, as I indicated, the year before when it was first cloned. And Leon was smart. We’d go down to the Petroleum Club, usually, which was an interesting experience for me—a five-foot-six Jew with the six- and seven-foot Texans coming in in their boots and all. I felt so out of place. I really did. I mean, I’m not saying there was any anti-Semitism, but it was just—I felt uncomfortable. First, I was a doctor. I don’t think I’m a nerd, but to them, maybe. And all these rich oil people with their—and they were smoking in those days, and I will come back to the smoking—the cigars and stuff—and, “How you doing?” and all that stuff. But we had had a private room, and Leon would set up this TV set, and he had the cassette that I don’t know where it is today, of the first McNeil/Lehrer of interferon being cloned and me being on there. You know, TV has a big impact, and that’s what sold it. It was that TV show.

After a while, I would start coming in late because I was busy, because I knew they were going to show the tape, and I didn’t want to watch that thing again—time and time again. It was kind of an interesting entrance. About halfway through, the man shows up, so to speak. I never thought of it that way. So John Bookout committed two million to me, and it just rolled from there.

I’m not going to go through all of them because I probably can’t remember right now, but we became—so they put together, on this board, some of the really leading people of Houston—Davis, Huffington, Bob Lanier—

[redacted]

So Lanier was at the thing. Ken Jamieson, who was CEO of Exxon at the time, Baine Kerr, who would president of Pennzoil, and a few other—quite a board. We would meet from time to time, but it would be Leon, me, and Roy who would go to Pennzoil, who gave a million, Atlantic Richfield in Dallas that would give a million, Cheney’s company—what’s the name of that one? It’s a drilling company, but it’s—forget it. I’ll think of it.

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I think almost everybody but one or two companies gave us either a half a million—and as Leon would repeatedly say in every single meeting, “We’re not here to raise—to sell Girl Scout cookies. We can’t do it at five dollar donations. We really want—we need a significant—.” But in those days—’78, ’79, ’80, ’81—the oil industry was rolling in profits as they are today. And philanthropy, I think, was easier then because there weren’t as many people doing it. Like I was probably—because I had this new idea, and this was sexy—genetically engineered protein. I mean, my God, I had a—you know. So it was highly successful, and we took that money. Literally not a penny went to anything but to buy interferon. They wouldn’t even pay for my nurses and stuff.

Then in 1980 a very important event happened. Leon took me over to a thing called—a foundation called the Clayton Foundation. I’m not going to have time today to talk about them, but they started giving me money for nurses and stuff. So I had a staff, because we couldn’t handle what was going on. I might just tell you that in 1995, after Mary died, I went to them with the idea I wanted to change fields. I had an abstract idea that plants a lot to offer, and we were missing big things in cancer. I thought there was going to be a revolution of how we understood cancer, and could I start working on a completely different—? It was an abstract—it was just like that paper there. It was just—I had no picture. I didn’t know what I was going to paint. I didn’t know what I was going to paint when I did that. I just let the emotions go. And they said, “Go for it. You did it before. We didn’t make any money. You didn’t make any money. Maybe this time we’ll get a patent. We’ll see.” We’ll talk about that story. But the Clayton people really helped me. So Leon played—and his wife played—a critical role here.

Now, I was having more and more—the last five minutes—more and more trouble here, though. There was a lot of backlash about all the publicity. I told you there was this thing about the hairy cell leukemia wasn’t real. And I had it up to here. So I got Huffington and Lanier, who hadn’t been mayor yet, but two very big, powerful Texans—I’m going to tell this story; it’s the way it happened—and Leon Davis. I set up a meeting with Dr. LeMaistre. And I didn’t blame him. I’m just saying that there are just too many roadblocks. Now, all of the sudden, people are stopping me.

I’m going to sidetrack for a second and give you the thing that just went over the edge. I got a memo around this time from the institutional review board saying, “Your original protocol where you had what is called Phase I interferon—you have now put five hundred and fifty patients on, and you cannot do that. You have to separate this out into disease types. The rules have changed. We’re going to stop you from ever treating another interferon patient with your natural interferon until you change the protocol.” I said, “We’ve already cured one disease with it. What are you talking about? What difference does it make if it is, on paper, five hundred patients or each is separate? You can’t stop me.” “Yes, we can.” That was it for me. I mean, that is what—I was—this was probably punitive, and it wasn’t fun.

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So the three of us—the three of them plus me went to see Dr. LeMaistre. And it was mainly Lanier who was the outspoken one saying, “Dr. Gutterman keeps saying there are roadblocks, and he just can’t seem to get the work done. It looks like he’s got the support of the administration.” All the right things were said. “What about this business of telling him not to go on McNeil/Lehrer, telling him because someone said it was fraudulent? What about that?” Well, finally, after about thirty minutes of this hemming and hawing, Lanier got up. He was so impatient with this because he was a business guy—he was—he’d do it. He stands up. He’s about six-foot-four. He smoked big cigars in those days. He goes right over to LeMaistre, who is sitting on this couch, and there was an ashtray there. He takes a deep breath—a big deep breathe—and he lets the smoke out. And he said, “Mickey, I just want a simple answer from you. Are you going to allow Dr. Gutterman to do his work or not? A simple yes or no is all I need.” “Yes, sir.” And he takes his cigar and—swoosh—all this smoke comes out. He said, “Gentlemen, have a good day,” and he just walks right out, all this smoking going—sort of like Woody Allen when he sneezed and all this cocaine was at a party and all this—I don’t know if you saw Annie Hall?

Tacey Ann Rosolowski, PhD

1:42:33.8

I did. Yes.

Jordan Gutterman, MD

1:42:34.2

He sneezed all the—a million dollars in cocaine goes up in smoke. So I saw through the haze LeMaistre was just kind of like—and I didn’t have a whole lot of problems after that. But it was near the end. It was near the end because it got approved and then Mendelsohn came in. And there were some other stories. I’m not going to get into them. One that—well, I know you’re interviewing LeMaistre, and I still—when he sees me, it’s very friendly. I think there is some admiration on both sides. I think he’s done a lot—he did a lot of good things. He had a tough time in accounting—we had some tough times here. I would say he always said the right thing, but I didn’t always feel I had the backing of the president’s office then. I did with Clark. But—you know—who knows? He was under some—perhaps a lot—of pressures and stuff. But when I see him, I have warm feelings about him. I think he was a good president. I mean, Clark was a great man. I think LeMaistre for the—he was here eighteen years. He did good things. I mean, we flourished. I did, for sure. And again, he kept the place such that—and Freireich had problems with him too. I don’t know if you asked Freireich that thing. Freireich probably told you a lot. Did he? I’m not getting into some stuff like Freireich got into. Yeah, that’s some tricky stuff. I’ve got to think about that.

Tacey Ann Rosolowski, PhD

1:44:03.2

Sure. Yeah, it is. It is.

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Jordan Gutterman, MD

1:44:04.9

I don't like to get into certain things, but it's part of the history.

Tacey Ann Rosolowski, PhD

1:44:06.3

Yeah. Well, I'm not necessarily looking for dirt and opening the closets, but an evaluation of how someone has operated in an institution—I mean—that's really what—you know—the baseline of what I'm interested in.

Jordan Gutterman, MD

1:44:21.5

Well, Freireich—I mean—LeMaistre hired this guy, [Irwin] Krakoff, and Freireich had a heart attack he was raging so much back in '85, I think it was. He had big problems, but he was more confrontational than I am. I tend to work around a problem. That's a good character—I mean—I tend not to be as confrontational, but he likes the controversy. I think Freireich thrives on that type of stuff. He likes chaos. He likes the confrontation more than I do. I admire that. I don't like that. In fact, I'm just the opposite. I tend to find my solutions in working around the problems in a positive way rather than—I would never have—but I kept complaining to Leon about I couldn't get my work done. It was somebody else you're interviewing that created a real problem for me. And I—whether I tell you about the story or not—but he blocked a grant to Clayton. And this somebody blocked the grant that you are interviewing—

Tacey Ann Rosolowski, PhD

1:45:22.4

Yeah. You spoke about it in one of your—

Jordan Gutterman, MD

1:45:24.1

Oh, did I?

Tacey Ann Rosolowski, PhD

1:45:24.6

Yeah, with Lesley.

Jordan Gutterman, MD

1:45:25.5

Did I mention who it was?

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1:45:25.8

Tacey Ann Rosolowski, PhD

1:45:25.8

Uh-hunh (affirmative).

Jordan Gutterman, MD

1:45:26.6

Okay. I'm going to keep that. I mean, he would deny it. Every time he sees me, because—you know—he is over in this building. He can't gush enough about seeing me. He loves to talk, and so he loves the old days. There's selective memory. He would probably never remember it, but he did hold that up. But that is kind of par for the course. Anybody who has done anything has had people who get in the way.

Tacey Ann Rosolowski, PhD

1:45:49.3

Sure. Sure.

Jordan Gutterman, MD

1:45:50.9

I actually like the guy. After a while, he became a great supporter. Once the stuff really proved that it worked, he became a great supporter. So I don't have any ill feelings. I don't have anything for LeMaistre either. Freireich probably does, but this is not about Freireich; this is about me.

Tacey Ann Rosolowski, PhD

1:46:08.0

We're at 3:20, so I want to make sure—

Jordan Gutterman, MD

1:46:09.3

Yeah, we better go.

Tacey Ann Rosolowski, PhD

1:46:10.2

Why don't we close off the interview for today?

Jordan Gutterman, MD

1:46:12.3

So—and the way—yeah—and I think we can talk about it or take it off either way.

1:46:15.3 (End of Audio Session 2)