Kwon-Chung, Kyung-Joo "June" 2018

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Dr. Kyung Joo Kwon-Chung (June) Oral History

March 26, 2018

Interviewer: Dr. Victoria Harden, Founding Director, Emerita, of the Office of NIH History and Stetten Museum

Dr. Kwon-Chun discusses her work as a mycologist in the Intramural Program of the National Institute of Allergy and Infectious Diseases (NIAID).

Harden: Dr. Kwon-Chung, you were born on August 16, 1933 in Seoul, South Korea, the oldest child in your family. Your father was a politician, your mother was a housewife. Would you tell me about your growing up and your early education, what interested you, and especially any experiences that might have directed you towards a career in science.

Kwon-Chung: Right. As you mentioned, I'm the first child in my family and my mother [Sung Ie Cho] was very serious about education first, an "education mommy" type of person. From very early on in my life, she was very serious that I become some professional career lady instead of simply a housewife. That was 1933. You can imagine that's 84 years ago. Most of the girls who were my peers were finishing high school, and some would go onto college, and then most of them would end up as ordinary housewives. My mother was very interested in making or at least training me into a scientist. She didn't exactly know what kind of field of science, but she just convinced me that I was born to become a scientist and then she would do everything to support my education.

Harden: What do you think motivated her to do this?

Kwon-Chung: I think that being the first child followed by another girl has a lot to do with it. In Korean society, especially then, maybe now, too, boys were preferred and valued more, unfortunately. She knew that I would be not only a daughter, but would function also as the one representing my family in the community. I think that was the reason. If I had an older brother, she might have had a different kind of idea about what kind of person I would grow up to be, but she was very interested in seeing me to grow up and become a scientist. From very early on, I wasn't interested in physical or chemical science. I was definitely more interested in biological science.

I was very curious and interested in human anatomy, and then birds and zoology in general but not so much botany. I knew vaguely that I would grow up to become a biologist from a very young age. My father [Choong Ton Kwon] was a politician. When I was very young, we were under Japanese control. He wasn't in politics until the second war was over. He was educated in Japan during the Japanese occupation, majoring in political science at Waseda University in Tokyo. It's natural that he wanted to eventually become a politician. After independence, which occurred in 1945, he was elected multiple times as a Congressman representing a south east district of the northern Kyunsang Province. He eventually became a defense secretary in the late 1950s to the beginning of 1960. I was growing up in this political family, and although we didn't want to be in the limelight, I was always recognized as "so and so's daughter," and what my father did was considered important. I think this environment made me feel like I must become a scientist that my parents would be very proud of, at least to meet their expectations. From a very early age, I had that sense of responsibility, as a first child, and then as the daughter of a very seriously education-oriented mother, and then as the child of a family who was already very much known in the whole area. I think those all played a role for me in deciding that I would be somebody in science eventually, rather than just a normal ordinary housewife. That idea came in from very early in my life.

Harden: You enrolled at Ewha Womans University--am I pronouncing it correctly?--Ewha Woman's University.

Kwon-Chung: Yes, Ewha Woman's University.

Harden: That was 1952 and the Korean War was on.

Kwon-Chung: Right. The war of course, was at its height when I was entering college. If you remember my college from the picture I sent, it had beautiful stone buildings. Our college was established by an American Methodist missionary group in 1886. It wasn't a college from the very beginning. It started just as a school for modernization of women. I don't know if it is correct to compare it with an elementary school in its early years, but it was a "learning house" for girls. Eventually it became a college in 1925—by the time I was entering, the largest college for woman in Korea and possibly in Asia.

The Korean War broke out in 1950. We were driven out of Seoul, which is where I was born and had gone to school and planned to go to college. Everything was there, in Seoul. It's the capital. We were driven away to the southern most city, and that's Busan. If you can imagine the temporary campus: multiple wooden barracks covered with the U.S. Army tents, which I remember were donated by the Eighth Army. We started our education in this temporary campus in Busan. You can imagine the quality of education we received, because there were no labs and no facilities. It was okay to learn English or history and so on, but not science. I say the Korean War was no less than a catastrophe for us.

Harden: The fighting ended withe the armistice in July '53. Did you get to go back to Seoul or not?

Kwon-Chung: We went back to Seoul but not immediately after the cease fire because it was still chaotic and the major part of Seoul remained completely flattened and destroyed. By the fall semester of my junior year, which started in September, we were allowed to go back and start at the Ewha University campus located in the outskirts of Seoul. The campus had been used as housing for the U.S. Army until the truce took place. I don't know which unit or division of the U.S. Army, but the campus had all been geared toward the needs of the Army, so it took time to get settled. So my science education in the first three years of college was minimal.

Harden: Lost, with respect to laboratory science?

Kwon-Chung: Yes. This is why I had such a hard time when I started my graduate work at the University of Wisconsin in 1961.

Harden: You did some graduate work in Seoul.

Kwon-Chung: Yes, that was a master's program for two years between '56 and '58. After that, I remained in Ewha as a teaching assistant, research assistant, and eventually attained, I guess it was a rank equivalent to an assistant professor. That's when I was given an opportunity to come to the United States.

Harden: What kind of research were you doing? I believe you were beginning to get into mycology at this point, were you not?

Kwon-Chung: No, my master's degree in Korea was about drug resistance in *E. coli*, and that was two years' work. I was more exposed to bacteria than to species of fungi until I came to the University of Wisconsin. There was no mycologist in Korea to speak of at that time, and during my college years, the professors at universities were not only giving lectures at their own universities but also giving lectures as a visiting professor at other universities twice or three times a week. They were rotating from university to university.

Harden: I see.

Kwon-Chung: Ewha University never had a microbiologist as a full professor and microbiology was always taught by someone who came from another university as a visiting professor, and there was no mycology course. By the time I became a research assistant and then began teaching microbiology at Ewha, I started to think about a possible future as a mycologist in Korea. I could see that there was a niche that I could fill since there was no mycologist at that time. I thought, "I'll go to the United States and study fungi, become a mycologist, and then come back to Ewha to teach mycology.

Harden: That's what you were thinking at that time?

Kwon-Chung: That's what I was thinking about until a coup d'état, a military coup occurred in 1961. At that time, my father was Defense Secretary. When the military coup occurred, you can imagine what kind of havoc ensued for our family.

Harden: Yes.

Kwon-Chung: Since my father was the Defense Secretary in the previous administration, he was briefly house-arrested, and our life was turned upside down during this traumatic period. At that time, a Fulbright scholarship was given to each university in Southeast Asia and Korea. I think they gave ten slots each or ten fellowships to each country, and the Korean government decided that they would fill this scholarship with junior college professors to be trained for one year in the U.S.—this was a one-year program for graduate work. What can you accomplish in one year? My university nominated me to apply for this Fulbright Scholarship in 1960.

I represented Ewha University and took an exam at the U.S. Embassy in Seoul. It was a written exam followed by an oral exam. I passed the exam and was awarded a scholarship. There were, I think, altogether seventy applicants who took the exam, and ten were chosen. I became one of them and was given the chance to come to the United States.

Harden: Before you came, however, I believe you got married?

Kwon-Chung: Yes, I got married during my master's program of two years. I already had two little boys by the time I was ready to leave for the United States.

Harden: That is just amazing to me, especially in Korean society. Your husband [Chung Young Muk] was happy to come to the United Stated with you?

Kwon-Chung: Not quite, because we didn't plan that way. It's interesting because he was also a son-in-law of a previous administration's Secretary of Defense and that affected his situation. In the previous administration, he did not have to be drafted into the army because he's the only son in his family and his father passed away when he was very young. His mother was already sixty by the time he reached draft age, and that time they had a special exemption from military service if a man was an only son of a widow more than sixty years old. When this military coup occurred in August--was it August? No, April, I think, April '61, then things changed. He had to go into the army because the previous law was no longer in force. He had to fulfill his military obligation. It was almost like there was no other option for us than me coming to United States and then we just go from there.

Harden: Your mother came with you?

Kwon-Chung: No.

Harden: No?

Kwon-Chung: She didn't come. She was in Korea and because this program was for one year only, she took over my kids for that year.

Harden: I see.

Kwon-Chung: She was taking care of my kids very willingly. She's an amazing lady of energy and stamina. She passed away four years ago, January 3rd, 2014, at the age of 101.

Harden: Amazing.

Kwon-Chung: She was one of those ladies that even three days before she passed away, she would read the newspaper from front to back. She had to know everything that's going on in the world. She took care of my kids, and I was supposed to come home after one year because this program just provided support for one year. But because of the Korean political situation, after I came to the United States, I said, "I'm not going to go back." As I said, my father was house-arrested, and things were just chaos. Before I left Korea, I didn't tell anybody, but I decided that I would pursue graduate work and finish a PhD. Nobody finishes a PhD in one year, so you need fellowships.

When I came to United States, and before I came I did some searching. I knew a visiting professor who was teaching plant pathology, and he just came back after a post-doctoral experience at the University of Wisconsin. He finished his PhD at the University of Michigan, Ann Arbor, and then went to the University of Wisconsin for post-doctoral fellowship. He finished his post-doctoral fellowship of three years or two years, which I don't remember, but he came back to Korea and was appointed to a professor position at one of the colleges near my home. I decided to visit him and get some advice before choosing a university. I said to him, "I'd like to become a mycologist because Korea doesn't have mycologists. Which school in the United States do you recommend me?" He said, "Oh, the University of Michigan Ann Arbor is excellent, but also the University of Wisconsin is very well known, and there is a professor called Kenneth Raper [Dr. Kenneth B. Raper], who is almost like a legendary figure in the genus *Aspergillus* and *Penicillin*." I said, "Okay, good idea." I sent my application for one-year training to Dr. Raper, knowing that he didn't have to worry about supporting me because the State Department was paying for my education and living expenses. He agreed to accept me. When I arrived at his office at the end of August, 1961, he said, "Okay, what do you want me to do? What can I do for you?" I said, "I'm here to work on my PhD." He said, "Not a PhD in one year. This program is for one year." Then he said, "Well let's not talk about the PhD at this moment. You're going to take this, this, and this course." He already had my curriculum laid out before my arrival.

It was the hardest struggle of my education, because, as I said, my undergrad science background was very poor due to the Korean War. I had no mycology background. Even my microbiology—I had some courses in bacteriology without any laboratory work and there was nothing on eukaryotic microbes or viruses. The course work was very difficult, and then there was the language problem. In the beginning, I may have understood 20% of the lectures at the most. I may have slept an average of four hours a day or something like that during the first few semesters. The introductory course in mycology was taught by Myron Backus [Dr. Myron Port Backus] of the Department of Botany. He was such a dedicated professor. His preparation for teaching material was beyond comparison with any other. He was so thorough. For example, if I had a one-hour lecture, the lab work was also supposed to be one hour, but we had to be in the lab at least three hours because he had so many materials for us not only to look at under the microscope but also to compare with the other fungi that were in the same class or even genus so that we could learn how to differentiate them. I mean he was thorough and absolutely superb. I had the best mycology education under his tutelage. This was an introductory mycology. I worked—I should not use the word “hard,” but if there is an adjective to describe “more than hard,” I would use that. I did my very best. When I received the first report card on introductory mycology, it was an A plus, and Dr. Raper said, "Now let's talk about your PhD."

Harden: Right.

Kwon-Chung: He had an NSF [National Science Foundation] research grant to write a new monograph of the genus *Aspergillus*. I had no problem getting support, and that's how I finished master's in two years and then went on to finish my PhD in a total of four years.

Harden: It was during your PhD work that you began to work on *Aspergillus*?

Kwon-Chung: Yes.

Harden: That was the focus of your dissertation I take it?

Kwon-Chung: Right.

Harden: Would you talk a little bit about what's interesting about *Aspergillus*?

Kwon-Chung: Well *Aspergillus* is the most ubiquitous fungi in our environment. You and I, even in this room, are inhaling at least an average of three spores of *Aspergillus* every hour. You cannot avoid them. The majority of them are saprophytic and they don't cause any disease because most of them don't even grow well at 37 degrees other than a few. Pathogenicity-wise, they are limited to few species. But because *Aspergillus* is so ubiquitous, it has industrial value like *A. oryzae* which is used to ferment soybeans to make a miso, and soy sauce. Other species of *Aspergillus* are used to make enzymes, organic acids, antibiotics, and other drugs such as cholesterol lowering Lovastatin. Dr. Raper was writing a monograph of the genus *Aspergillus* in the 1960s and wanted to include as many species as possible.

When I got onto this PhD program, there was another graduate student in Dr. Raper’s lab who was studying cellular slime mold, which is a soil organism like *Aspergillus*. He was going to tropic/subtropical areas like Costa Rica and other places collecting soils from which to isolate slime molds, and then the same soil samples were given to me to isolate *Aspergillus*. I was isolating *Aspergillus*, one after another, and I forget how many new species I have discovered. They were all included in the monograph *The Genus Aspergillus* published in 1965 by Kenneth Raper and Dorothy Fennell.

Most interestingly I discovered something new--of course, I stumbled on it, as I didn't know what I was doing at that time, because all I knew was that I was supposed to be isolating *Aspergillus* and describing their morphology and how they grow. But in a Costa Rican soil sample, I isolated *Aspergillus* species that mates. This was the first known heterothallic *Aspergillus* species, meaning that mating between two compatible strains is an obligatory process to complete the sexual lifecycle in this new species. At that time, there were probably about 100 species of *Aspergillus* known, and this was the first heterothallic species in the genus *Aspergillus*. I discovered it from Costa Rican soil, and it posed the question of how to describe it, because you don't have a female and male situation. I called it mating type “small a” and the opposite mating type as “large A.” Interestingly, one mating type strain was orange in color and the other mating type strain was yellow in color, the colonies of the organism. It was beautiful to look at because they were aesthetically pleasing. I was really absorbed in deciphering the lifecycle of this *Aspergillus* species to see whether heterothallism was controlled by one gene with two alleles or whether it was more complicated than that. This became my thesis material.

Harden: If I may ask, fungi are much more complicated than bacteria or viruses. Why is it that they were not looked into more early on? Was it because people did not realize that there were that many pathogenic fungi?

Kwon-Chung: I think that has a lot to do with Pasteur and Koch’s accomplishments. They worked on bacteriology, and as bacteriology made huge progress , mycology was overshadowed.

Harden: Yes.

Kwon-Chung: Actually a fungal study about which you may be interested is the one that fulfilled a Koch postulate with a fungal pathogen at least 40 years before the works of Pasteur and Koch on bacteria.

Harden: Interesting.

Kwon-Chung: This was with ringworm fungus. Dr. Gruby [Dr. David Gruby] found that this mold caused ringworm disease, and he proved that the mold was the cause of the disease. He was the first one to prove what we call Koch’s postulate linking a particular organism to a disease. Because fungi causing ringworm disease didn't kill, they were mostly studying dermatophytes that cause skin disease and so on. They were nuisances and ugly, but they were not considered as serious as what Pasteur or Koch were studying.

Harden: Tetanus. Diptheria. They were so dramatic.

Kwon-Chung: Yes—their seriousness and death rate and so on got much more attention. I think that mycology was very much overshadowed by bacterial disease, and progress in bacteriology went way ahead of mycology.

Harden: In 1966, you came to NIAID as a Fogarty International Fellow. Chester Emmons [Dr. Chester Emmons] brought you here because your expertise on *Aspergillus*. Let's step back one step. Did your family in Korean come when you came to NIH or was it later?

Kwon-Chung: Oh, they came after I came to NIH. My husband arrived in 1963, and by 1966, we had a third child, who was born in Madison, Wisconsin, in 1964. I was pregnant while finishing the PhD. I took my final exam, I got the PhD in August 1964, and my daughter arrived on October of that year.

Harden: I'm laughing because I had a post-doc who had a baby about one month before we got a book out on which she was the principal author and editor. Women somehow are able do this! Go ahead.

Kwon-Chung: We moved to Washington in 1966, and two years later, the boys arrived, and then my mother joined us, so we were all reunited.

Harden: Returning to your work at NIH, I believe that you came to NIH, NIAID, because of your expertise on *Aspergillus*.

Kwon-Chung: Actually, not *Aspergillus* specifically but because of my knowledge of fungal sexuality. The obligate mating of fungal species, to complete the lifecycle, I was the first one to discover that this happens in the genus *Aspergillus*. At that time, actually almost at the same time, Dr. Stockdale [Dr. Phyllis Stockdale] in London was discovering that ringworm fungi also has this type of sex and heterothallism. Dr. Emmons was keenly aware of that. When I applied to him, I said, “I have studied the sexual lifecycle of *Aspergillus heterothallicus*. I named it *Aspergillus heterothallicus*.” I also said that when I am accepted by NIH, I would like to study heterothallism in ringworm fungal species, whether genetic control of their sexual lifecycle is by one gene/two alleles or two genes/four alleles--whatever the genetic system may be, because it was not known then.

He said, "Of course you can come," because the study of heterothallism in pathogenic fungi was something very new and an unknown area. He needed somebody with expertise in it. He didn't have to come up with a new project for me. I said, "I'm going to do this," and so he said, "Come over and do what you plan to." I studied the sexual state of the fungi causing Dermatophytoses. Those are my first publications in *Sabouraudia*, which later changed its name to *Medical Mycology.*

Harden: Would you describe the lab, Dr. Emmons lab when you arrived. Who all was in it? What problems in general were they investigating?

Kwon-Chung: When I arrived at Dr. Emmons lab, I was the only female scientist. Dr. Emmons was ready to retire, which I didn't know. He never told me that he was going to retire six months after my arrival. I had planned to learn about pathogenic fungi, because my training was in general mycology, about the fungal species that are innocuous. I wanted to learn about fungi that cause disease, and starting with ringworm, that means cutaneous disease, and then eventually I wanted to go into the fungal species that cause systemic disease. I was just hoping that I would learn all these things from Dr. Emmons, who was a pillar of medical mycology in the United States. He was the basic research pillar of medical mycology. The clinical pillar of medical mycology was at Duke, Dr. Norman Conant [Dr. Norman F. Conant]. Two of them were really the pillars supporting the medical mycology field in the United States.

Anyway, when I arrived at NIH, Dr. Emmons was mostly gone to Peru and other countries in South America for workshops and so on. I almost never saw him. It's like, okay, here you are and you became an orphan because I didn't have a mentor. It was a good thing that I said I want to study the heterothallic lifecycle of the *Dermatophytes* and decipher genetic factors that controlled their reproduction. If I didn't have a project already identified, I don't know what I would have been doing, since there was no one to lead me or mentor me. Anyway, after I arrived, Dr. Emmons said that he was planning to retire, and most of the time he was in South America giving workshops. It wasn't the environment that I expected. I thought that I was going to have daily interaction and learn from Dr. Emmons and then get new ideas about how to move on to other fungal species and their pathogenicity, but the training I needed was not available.

I was lucky that Dr. Jack Bennett [Dr. John E. Bennett] was in the clinical mycology section at the LCI--at time they called it LCI, the Laboratory of Clinical Investigation. He was a senior investigator there. He was someone that I could fall back on.

Harden: He had training in mycology?

Kwon-Chung: First of all, he's a very strong, absolutely a most famous infectious disease person now, and even then, when he was young, he was not only a good infectious disease person, but he was studying *Cryptococcus*, and his main interest was in fungal pathogens. At least one person in the clinical area at NIH was interested in fungal pathogens. We got together once in a while and exchanged the histology tissue sections of some fungal disease so I could learn what the fungus looked like in human tissue, because all I had studied were fungal cells *in vitro*. It was great that Dr. Bennett was there. Otherwise, I would have never had any opportunity to access human clinical specimens.

Harden: What were the other people in Dr. Emmons lab doing? Were there a lot of them?

Kwon-Chung: No, there were two or three technicians who helped senior investigators, and then there were two senior investigators: Dr. Herbert Hasenclever [Dr. Herbert F. Hasenclever], who was a *Candida* person. Dr. Lonas [Dr. George Lonas] studied dimorphism of *Coccidioides immitis*. So there was one person who studied *Coccidioides*, another who studied *Candida*, and then I came to work on the *Dermatophytes.*

Harden: They were not interested in the clinical manifestations of what they were studying?

Kwon-Chung: No, not really. They were doing very basic studies. Dr. Lonas was interested in how in the parasitic phase, so-called spherules are formed *in vivo*. When people inhale the etiologic agent of coccidioidomycosis--that's arthrospores--the inhaled saprophytic conida convert to huge spherules and produce numerous endospores within the spherule. How does this organism convert from the mold stage (saprophytic atage) to spherule (parasitic stage) stage by completely changing its morphology? He wanted to see what triggers, biochemically, the initiation of morphogenesis. At that time there was no DNA, no molecular genetics. So they were investigating biochemically the kind of growth environment that enhances the transformation of mycelium to spherules in vitro. It was a very classical biochemical study. Then Dr. Hasenclever was studying the antigenicity of *Candida* cell walls. He was discovering the serotype--there are two different serotypes among *Candida*, serotype A and B. That's an antigenic aspect. Three of us were working on very different aspects of pathogenic fungi.

Harden: When you started working with Dr. Bennett and getting into more clinical work, you were working, if I am correct, on *Histoplasma*?

Kwon-Chung: The work with *Histoplasma* was before I joined Jack Bennett because it was during '70 and '71. Remember, I came with the background of fungal sex, heterothallism. I was wondering, maybe *Histoplasma* has a heterothallic sexual life cycle. Actually, before I had that interest, someone published that *Histoplasma* had a sexual stage, and the fungus produced a sexual stage, but that it was not heterothallic. I said, "There is something not very clear about this report and I'd like to look into it myself." We had quite a collection of *Histoplasma capsulatun* strains that Dr. Emmons had collected. I pulled those out. Also, I noticed that in Kansas there was a CDC [Centers for Disease Control and Prevention] branch where studies on ecology and epidemiology of pathogenic fungi were carried out. One person there was collecting soil samples where *Histoplasma* had not been known to present. He had quite a bit of soil samples, which I requested, and I received the soil samples from him. I isolated numerous strains of *Histoplasma cap sulatum* from the samples and then started matchmaking to see if the fungus is heterothallic. I found that *Histoplasma* has a heterothallic sexual cycle, and I published it, I believe, in 1971.

Harden: I saw it in *Science* in 1972.

Kwon-Chung: Yes, '72. That's when I named the fungus *Emmonsiella* to honor my professor, Dr. Emmons.

Harden: Yes. It was a new species, Kwon-Chung.

Kwon-Chung: Yes, because during that time, the fungal taxonomy was based on the sexual state, and the asexual state was what we called a Fungi Imperfecti or Deuteromycota. It's almost like you collect all these species that do not produce a sexual state in a bag, and they call it Deuteromycota or Fungi Imperfecti. The sexual state was called the "perfect state." Once the sexual state was discovered, you give a new name for the sexual state and classify it in the appropriate position in systematics. We then know which class it belongs to, because the division or classification in the fungal kingdom was based on the morphology of the sexual state. When the sexual state was not discovered, it was put into a sort of storage bag with all the others that did not have a sexual state. Once we discovered the sexual state, it was taken out and placed into its correct phylum and order, a correct kind of a classification. You give it a new name, and I gave the name *Emmonsiella capsulata*, keeping the species name the same but giving it a new genus name. This is how *Emmonsiella capsulata* Kwon-Chung sp. nov. came to be.

Harden: I want to step sideways for one minute, given our discussion of this and note that when we were given information for that anniversary book for NIAID about organisms named after people, we got Chester Emmons with this name, but your name was not there. It would seem to me that the full name includes Kwon-Chung sp.Nov new species, right?

Kwon-Chung: Right. *Emmonsiella capsulata* Kwon-Chung, species. nov.

Harden: One would think that your name should be on the list of NIH scientists after whom organisms have been named. Why was it left off when we published a list in the 1987 *NIAID Intramural Contributions* book? I'm just wondering if this was a typical male bias in compiling the list.

Kwon-Chung: No. I think that the person who discovered a new species is the author of this new species. The correct citation would be *Emmonsiell a capsulata* Kwon-Chung 1972. A few years afterwards, when the phylogeny and DNA sequencing and this kind of a thing came in, someone put *Emmonsi ella* into preexisting genus *Ajellomyces*. That means its name now is *Ajellomyces capsulatum* (Kwon-Chung) McGinnis & Katz. Whoever named the *Ajello myces* comes first. However, rules in Fungal Taxonomy have changed since 2003, and the first name given for the fungus, *Histoplasma capsulatum*, is the name now used.

Someone did name a new genus of yeast after me. There is the genus *Kwoniella*, and that's yeast species discovered by Dr. Fell [Dr. Jack W. Fell] at the University of Miami. He is a marine biologist, but his expertise is in the yeasts that belong to *Basidiomycetes*. He has described tons of new species and new genera. One day he called and said, "I'm going to name a genus after you." He had discovered a new genus. He wanted to use my full name Kwon-Chung. I said, "Why don't you just use the Kwon," because Chung is my married name--got hooked up with Kwon when I married--so you can just call it *Kw oniella* instead of using my full name of Kwon-Chung.

Harden: Do you remember what year that was?

Kwon-Chung: It was 2012. There are now several species in the genus *Kwoniella*. He discovered the one species first, but then several species are now recognized.

Harden: Your work on *Histoplasma*, finding the sexual state, helped to secure your promotion to senior investigator in the Laboratory of Clinical Investigation. Dr. John Seal was NIAID Intramural Director at that time, I believe.

Kwon-Chung: Dr. Seal, I remember. I was supposed to be put onto a more permanent kind of position because when a post-doctoral fellowship is over, then you have to become a principal investigator or leave. Dr. Seal wanted to support me. He wanted to put me into LCI as a senior investigator, but at that time President Johnson [President Lyndon Johnson] had instituted a hiring freeze starting from July 1, and my paperwork was not finished until after June 25, so there were not many days for it to go from one desk to another to another to be signed off on. It was a hot day, but Dr. Seal carried my package from Building 5, where his office was, to Building1, then on to Building 31 to get the necessary signature on the same day so that the paper work would meet the deadline before this hiring freeze on July 1st. This is why I moved from being a post-doctoral fellow to the next step on June 28, 1968. If he had not walked the package around--if he had just used the campus interoffice mail—I would not have made it.

Harden: That's the kind of boss you appreciate having.

Kwon-Chung: Oh, I will never forget Dr. Seal's special effort. I mean he wasn't a mycologist, nor did he have a special interest in fungi, but he was definitely interested in protecting me and my job and in supporting my career here. Otherwise, why would he care about whether there was a hiring freezes or not? I wasn't even a U.S. citizen at that time, so it was more complicated to hire me. There was no H-1b visa then.

Harden: When did you become a citizen?

Kwon-Chung: I think it was 1972 or '73 or something like that. Because from 1974 on I was on the government retirement system, so I became citizen before 1974.

Harden: In 1975, you began publishing on *Cryptococcus*, the study of which became 75% of your career's focus. Tell me what brought you into *Cryptococcus* research.

Kwon-Chung: As I said, my background is fungal sex, and I was asked why I was interested in fungal sex. The thing is that time there was no DNA, remember.

Harden: That's right.

Kwon-Chung: The only way to study fungal genetics was to mate fungi and analyze the progeny. Now you have a phenotype, your genetic marker, and you analyze how it segregates, whether it segregates or not. Whether one gene is involved or multiple genes are involved, this kind of thing, without this recombinational analysis system, you couldn't study genetics. This is a classical genetic study. To open up a field of genetic study, you have to find this lifecycle, where the mating system had to be discovered .

This is why when I discovered the *Histoplasma* sexual state, I wanted to study *Histoplasma* genetics. But *Histoplasma* are notorious in losing the sex so quickly when you keep them on an agar media in a test tube or another media in a laboratory. Not only that, the *Histoplasma* lifecycle took very long to complete. I thought, “This is not an ideal organism to study or to build my research program around for a long time.”

In contrast, *Cryptococcus* is a single cell budding yeast. They multiply and produce a colony very quickly. Within seventy-two hours you can see a colony already formed. At that time also, there was some indication that *Cryptococcus* may have a sexual state. There was an investigator called Jean Shadomy [Dr. Jean Shadomy], who was also in the NIH Clinical Center, in LCI, who discovered that some strain of *Cryptococcus* isolated from a human lesion produced a hyphae.

Harden: What are hyphae?

Kwon-Chung: *Cryptococcus* is just a budding yeast. One of the criteria to tell the genus *Cryptococcus* that time was that they do not produce hyphae. It should just be a budding yeast. That was almost a hallmark of this genus, and no one observed hyphae production in *Cryptococcus* prior to Dr. Shadomy’s description.

Harden: What are hyphae though? I'm sorry.

Kwon-Chung: Oh, a hypha (plural, hyphae) is a filament produced by fungi.

Harden: A filament, okay.

Kwon-Chung: Yes, filament with septation (walls that separate the cells in the filament). This was a completely different morphology. It was a filament versus a budding yeast. She discovered the strain that produced a filament, and that was isolated from a patient who had osteomyelitis on his hip, and that was Jack Bennett's patient. Dr. Bennett saw hyphae formation in this strain and he said, "You study this. I'm not interested." At that time, I wasn't there, but I heard the story that he noticed something interesting but that he didn't pay any attention. Jean Shadomy took over the strain, and she studied the hyphae formation. She discovered that this hyphae had a structure called clamp connection.

Harden: What does that mean?

Kwon-Chung: “Clamp connection” means that a sort of a clamp is produced from each septum to fuse to the adjacent cell so that there can be a nuclear transfer from one cell to the adjacent cell. It's a bridge, kind of. This is also a hallmark of Basidiomycetes, fungi that produce spores that grow out of microscopic cells called basidia. In the kingdom fungi you have several different phyla and the most advanced and complicated phylum is the Basidiomycetes. That's where mushrooms, puff balls, and all these jelly fungi belong. Those are macroscopic--you can see them--but there are numerous microscopic ones, too. Basidiomycetes fungi are the only ones that produce clamp connections when they produce hyphae.

We called this cryptococcal strain “NIH 12” because it was number 12 in Bennett’s collection, and it was producing clamp connections. Jean studied it for two to three years, and just observed up to the clamp connection production. I thought, “Well, once a clamp connection is there, that means it is Basidiomycetes. That means very likely this has a sex. The only reason why it has never completed and produced spores, is because it didn't find the right mate. Maybe its heterothallic.” That was my assumption. I asked Dr. Bennett, "Can you pick out twenty clinical and environmental isolates for me to play around to see if they have a sexual state?" I would start matchmaking, and without knowing that in *Cryptococcus* one sex--one gender--is predominant over 99% of the isolates. The other mating type is such a minority, you definitely have to be lucky to find it. Well, when he gave twenty strains, there were two strains that came from Denmark, isolated from pigeon droppings in the environment, which were mating type a, which is very, very rare.

I didn't know that; I was just culturing them in all possible combinations. One day I saw not only completely fused clamp connections, but spores being produced on top of the basidia, and I couldn't believe my eyes. I called Jack Bennett and said, "Jack, do you see what I see under this microscope?" I still have that microscope. He looked at it and said, "My goodness, I've never seen this. What is it?" I said, "This is the sexual state of *Cryptococcus*. Obviously the sexual state. Now I'd like to really delve into *Cryptococcus,* not only its lifecycle but also its genetic control, physiology, pathogenicity and all that." This is how I stayed in the *Cryptococcus* field.

See how luck plays a big part? If there was no *MAT*a (mating type a) strain among the twenty strains, I wouldn't have discovered it, and it is possible that even now we wouldn't know.

Harden: By 1975 and '76, you were beginning to receive awards for this work on *Cryptococcus*, and those awards I think may have spurred on your research to create a genetic test to confirm the virulence factors. Would you tell me about this work?

Kwon-Chung: Once I completely figured out the cryptococcal lifecycle, then I said, "What's next?" I figured out that there is one gene, two different alleles that control the sex. Then what? Now you have a genetic system, so you can open up a genetic study. First of all, you would like to study why this species is the only one of so many species in the genus *Cryptococcus* that causes fatal disease by disseminating from lung to the brain and meninges. Cryptococcosis, if you don't treat it, is 100% fatal. I said, “What do I look for?” Of course, I wanted to look into the role of the capsule, since people have already irradiated cryptococcal cells with UV and made an acapsular strain and then injected it into mice. Then they said, "Well this is a virulence factor because once crypto loses the extracellular capsule, it becomes no longer pathogenic."

At that time we knew well about the *Pneumococcus* capsule and that the capsule is an important virulence factor, not only in fungus but in bacterial systems also. But every species in the genus *Cryptococcus* has a capsule. Why is *C*. *neoformans* the only one which is virulent? I said, “Besides the capsule, there must be some other features that make this species virulent.” I was looking at the *Cryptococcus* character. Two things stood out to me. One was that this was the only species that readily grows at 37 degrees. The rest of the *Cryptococcus* species at that time were known to grow, at most, 35 degrees, but none grew at 37 degrees.

Harden: And 37 degrees Centigrade is human body temperature.

Kwon-Chung: Yes. That makes sense, right? Then the second thing that stood out to me was that this was the only species that produced melanin when you grow them on diphenolic substances. This phenomenon was discovered at the Robert Koch Institute in Berlin by Dr. Staib [Dr. Fritz Staib]. Also, everyone knew that Chester Emmons had discovered that *Cryptococcus* is associated with pigeon droppings. It's proven worldwide that pigeon droppings are the primary environmental niche of *C. neoformans*. Since pigeon droppings are a major source of *Cryptococcus neoformans,* Dr. Staib thought that maybe pigeon droppings are rich with cryptococci because pigeons eat bird seed (because people feed them bird seed), and the bird seed may have some kind of compound that enhances the growth of cryptococci. That's what he was thinking. He bought the bird seed *Guizotia abyssinica* [commonly known as niger seed]. He bought a bag full of them, ground them up, and concocted an agar media. We call it bird seed media. Then he grew the clinical yeast C*andida,* and it produced a white colony as on regular media. Then, when he grew *Cryptococcus neoformans,* it was the only one that produced a melanin colored colony. He said, "*Cryptococcus* produced melanin on this bird seed agar," which he called Staib agar, after his own name.

Before he discovered this, how did we confirm that a clinical isolate is really *Cryptococcus neoformans* and pathogenic? To confirm it, we needed more than the presence of a capsule, because every *Cryptococcus* has a capsule, and then also 37 degree growth? Yes, they should grow, but then some which grow at 35, and so how do we know that skin infection (skin temperature is 35 degree) is not due to some other species than *C. neoformans*? To confirm the presence of a cryptococcal pathogen, you inject the yeast cells into mice and confirm that mice die from cryptococcosis. Only then did you have a confirmatory diagnosis for *Cryptococcus neoformans*. You can imagine how many resources were needed to diagnose one case of *Cryptococcus* inf ection.

Because of his discovery of melanin, diagnosis of *C. neoformans* became simple because mouse study was no longer needed. He was assuming that bird seed has a compound that *Cryptococcus neoformans* turned the color. What was the compound? It was a diphenolic compound. We knew there are so many polyphenolic compounds such as caffeic acid and neurotransmitters like dopamine, norepinephrine, epinephrine that are substrates for cryptococcal melanin formation. When you add these compounds into agar and culture *C. neoformans* cells, they become dark brown (melanin color).

I said, "This is the only species in the whole genus having melanin producing ability. There has to be some reason why.” I started making a mutant, which lost the melanin, so its colony was white on bird seed agar. I crossed it with the wild type strain, which makes melanin. I got the progenies of both white and dark, so I knew that melanin production was controlled by one gene because the ratio of white and dark progeny was 50/50, a textbook case of Mendelian inheritance. I picked a few white progeny and a few of the melanin positive progeny to study their virulence in mice and it was completely clear that melanin is an important virulence factor. But people could still say, "How do you know you didn't mess up other genes by UV radiation? How do you know that you had mutated the gene that controls melanin production only? You may have introduced mutation in some other genes.” Cloning and other methods would have been definitive, but at that time, they were not available.

Harden: You were working on *Cryptococcus,* which is 100% fatal if it infects the brain, but no one thought of it as a much of a threat to humans. But after 1981, when HIV/AIDS appeared, *Cryptococcus* infection became an AIDS-defining disease. In 1983, you were on that paper with Tom Folks [Dr. Thomas Folks] and Ken Sell [Dr. Kenneth Sell] that reported the isolation of a fungus that grew well from the blood of AIDS patients. That's the paper, if I recall correctly, that people liked to laugh at after the causative retrovirus was discovered. I read that paper, and you all noted that this finding was preliminary and might not hold up.

Kwon-Chung: Not only that, we didn't say this was the etiology of AIDS.

Harden: Tell me about what this group of people were thinking about. My understanding of the period is that no one knew what the cause of AIDS was, so people were looking at everything possible.

Kwon-Chung: I wasn't so much interested in AIDS itself, because even the time that I discovered the heterothallism and published, the association between *Cryptococcus* and AIDS wasn’t peaking. I wasn't thinking much about AIDS. I was interested in *Cryptococcus* itself. One day I was in the lab, and a research fellow or clinical fellow, I don't remember, I even forget his name, marched toward my lab, and he was so excited. He said, "June [Dr. Kwon-Chung’s American nickname is “June”], we have this sample here. This must be something very interesting." I said, "What is it?" He said, "This is from an AIDS patient who came from New York, and we were separating macrophages from his blood." Then there was another patient, not from New York but from somewhere else. When they centrifuged his blood to collect the solid cells and separate out the macrophages and so on, he had the same fungus as the earlier patient, *Thermoascus crustaceous*. This fungus was not a common air contaminant that you usually see. I have never in my life seen lab air contaminated with *Thermoascus crustaceous*. If it's *Aspergillus*, I could say, "Ah, that's a lab contaminant," but this fungus I personally had never seen. Dr. Sell, the NIAID intramural director at that time, said, "What does this do?" He was very much interested and so excited to tell me about this. I grew the fungus, and I looked at its characteristics, and it was *Thermoascus crustaceous*. I decided that's the correct speciation. We said, "This came from two unrelated patients, and came from the blood source," but never said that this was the etiology of AIDS. Not many people paid attention, number one, but those people who were looking at AIDS etiology said, "Oh no, no, no. It can't be a fungus. Fungi don't do this kind of thing." People may have laughed, but we never claimed etiology for the AIDS.

Harden: People jumped to all sorts of conclusions. I won't go on about this. In 1985, you realized that the revolution in molecular biology that had been growing through the 1970s and into the 1980s meant that you needed to retool to continue your research. Tell me about deciding to do this and the course, the three-week course you took at Cold Spring Harbor Laboratory.

Kwon-Chung: If you remember when I said, “Melanin is a virulence factor,” then some people said, "How do you know you didn't cause another mutation by UV radiation? This melanin may be just an incidental association but not really the cause.” What is the solution then? You have to clone the gene. Clone the gene which controls the production of melanin. You have to knock it out or cause a mutation, and then see that the mutant becomes no longer virulent. Then you put the wild type gene back and the strain recovers virulence. It's like Robert Koch's postulate.

To be able to do this, you can't stay away from molecular biology. The handwriting on the wall became clearer each year. Actually it started in the early ’ 80s, and the message was, "Without molecular biology, you're not going to sustain your place in this scientific field. Either you retire or move into administration, or if you want to stay in basic science in the laboratory, you have to move on to reorient yourself." Well it wasn't easy. I was already fifty-three years old. I thought, "How do I re-orient myself?" I already had a twenty-five year career, and so I said, "Well, I have to do this." I was looking at a course that I could take without much sacrifice for my family and the lab, like leaving country or something that took months and months. I still had little children at home, and even though my mother lived with us, without me it would have been a major impact for my family.

I decided to take the Cold Spring Harbor yeast genetics course.
They were teaching us the fundamentals of molecular biology. What we learned was how to isolate DNA and how to run the CHEF (Contour-clamped Homogeneous Electric Field electrophoresis) to separate chromosome bands. Never mind gene cloning--we didn't get there. This was helpful for my psychological orientation, so that I could say, "Yes I can do this, and I need to do it." While I was there, I isolated a mutant without any chemical mutagenesis or UV radiation, just by selecting a spontaneous mutant which cannot synthesize uracil. When I came home, this mutant became a tool that allowed us to develop a transformation system in *Cryptococcus*. I collaborated with Dr. Jeff Edman at the University of California, San Francisco, who had been trained in molecular biology. We transformed the uracil mutant I isolated at Cold Spring Harbor Laboratory in 1985, using the electroporation system. This was the first successful experiment in inserting foreign DNA into *Cryptococcus neoformans* to transform.

Harden: In the years following, you expanded into publishing on the molecular genetics of several different fungal organisms. Are there any other things about retooling your research efforts that came out of this big shift?

Kwon-Chung: Taking a three weeks course doesn't make anyone a competent molecular biologist. You need someone who is very good at cloning, deleting genes, and other molecular technology. I said in my mind, "I need a person, my right-hand person. I can give the idea, but he can do the work because he's good in technique." I was thinking about who would be suitable for this lab. I was figuring out who was training fungal molecular biologists. At that time, Dr. Bill Timberlake [Dr. William E. Timberlake], who used to be a bacterial molecular biologist, had moved into studying the molecular biology of *A spergillus* morphogenesis, and I thought that maybe he had a post-doctoral fellow who was ready to leave his post-doctoral position and get a real job. One day I called Bill and said, "Do you have anybody who's ready to leave?" He said, "Yes, we have someone who is ready to leave." That was Yun Chang [Dr. Yun C. Chang], a staff scientist who has worked with me for more than twenty years now in our lab. He was the right person, so when I said, "Let's go after the *Cryptococcus* capsule, clone the genes that are responsible for producing the capsule, and then when we do that, let's prove that this is the virulence factor by fulfilling molecular Koch's postulate." This is how we were the first ones to be able to fulfill the molecular Koch's postulate on fungal virulence factor with the cryptococcal capsule.

Harden: Amazing. In 1995, you were named chief of the Molecular Microbiology section in the Laboratory of Clinical Immunology and Microbiology. I think that Tom Kindt [Dr. Thomas Kindt] was your intramural director at that time. In 1996, you received multiple awards, the NIH Director's Award, the Korean Overseas Compatriots Prize for Science, the Medical Mycology's Rhoda Benham Award. Indeed, from this time on you receive many, many awards that culminated with your Lifetime Achievement Award from the American Society of Microbiology. In a lecture you gave when you received that award, you identified four areas in which your career had fostered advancement in mycology, and we've covered most of these as you've talked about your work, but I would like you to give me a brief summary of these four areas and what kind of impact they made.

Kwon-Chung: One of these areas, certainly, was mycological taxonomy, because as I

said, many species were in a “bag” called Fungi Imperfecti. *Cryptococcus* was one of them. By discovering the sexual state that belongs to Basidiomycetes, this fungus has now been placed into the correct phylum, Basidiomycota, and its sexual state is now placed in the genus *Filobasidiella*. I named the genus *Filobasidiella* to accommodate the species *Cryptococcus neoformans*.

From there, I discovered that the heterogeneity among *Cryptococcus*, serotypes A, B, C, and D had already been discovered before I came on and the serotype had something to do with species identity. Dr. Bennett had screened every one of his stock culture with the antigen-antibody reaction and identified serotypes A to D among them. When I discovered the sexual state, I discovered two different morphologies, depending on the serotype. Serotype A and D belonged to the same group, and B and C belonged to the same group. I said, "There is heterogeneity, not only antigenic heterogeneity, but there must be genetic heterogeneity and there must be multiple other kinds of differences.” This is how the taxonomy field has been moving. Now there are many, many cryptic species recognized within the *Cryptococcus neformens* and *C. gattii* species complex. My original discovery of the sexual state of *Cryptoccus neoformans* ignited that field.

A second contribution to advancing medical mycology involved identifying the virulence factors of *Cryptococcus* and then transformation system making the technique possible with *Cryptococcus*, so this was a contribution to molecular biology. I humbly should say that we laid the stepping stone for other people who will come into the field.

Then melanin. Melanin research has moved very fast, and Peter Williamson [Dr. Peter Williamson] is now completely into immunology with all kinds of subjects, like apoptosis and autophagy that have stemmed from his experience in cryptococcal cell biology starting from genes associated with melanin synthesis and secretion. He actually started with the biochemistry of melanin synthesis. Then he looked at how many genes are involved in melanin production, regulation of production or secretion or translocation of the intracellularly produced melanin into the cell wall. He has revealed the entire pathway during the past 25 years. His study on melanin synthesis in *C. neoformans* became the base from which his study moved onto the next stage, and he was expanding greatly. So, I should say that melanin and the capsule as the virulence factors laid the foundation for further studies of virulence factors.

Harden: I was impressed with the Ecology of Epidemiology . . . .

Kwon-Chung: Oh yes, ecology, because we already knew that serotypes A and D of *Cryptococcus neoformans* came from pigeon droppings and soil contaminated with the pigeon droppings, but we never could identify the source of serotypes B and C strains, which I described as a different species, *C. gattii*. We had the pigeon droppings from California and Florida because as it turns out, these serotypes are common in tropical and subtropical regions but we couldn't find them from pigeon droppings. Not only do the sexual spores produced by serotype B and C strains look different, with spores shaped like fingers and not round like the spores that *C. neoformans* (serotype A and D) produces, they were not found in pigeon droppings. They are morphologically different, antigenically different. So, the question was, “Where are they in the human environment?” People also started paying attention to these two different species and they kept on finding more and more difference between these two species.

One is that 70%, 80% of serotype A and D, *C. neoformans* strains are isolated from HIV-positive patients. AIDS patients are at the highest risk for cryptococcosis due to serotype A and D strains. When you look at the patients infected by serotypes B and C, a lot of them are so-called non-immunodeficient people or normal people, without underlying immune deficiency. Serotypes B and C can still infect AIDS patients, but more so-called non-immunocompromised patients get cryptococcosis from the B and C serotypes. How do we locate where they are coming from? We have to know where the major environmental sources of *C. gattii* are.

We studied the biochemical difference of serotype A/D versus B/C. We noticed that serotype B/C has a very different way of utilizing a nitrogen source. We noticed that--some other investigators found--the strains of serotype B and C can utilize D-proline (a D-amino acid) as the sole nitrogen source for growth, while serotype A and B strains of *neoformans* cannot. We knew the D-proline utilization was not perfect for distinguishing *C*. *neoformans* from *C. gattii* because of false positive reaction among *C. neoformans* strains. We were testing all those amino acids, and we found that glycine, the simplest amino acid, plus L-canavanine in agar with bromthymol blue, which is a pH indicator, could differentiate the two species without false data. When the organism utilizes glycine as the nitrogen source as well as the carbon source, then they grow on this media and produce ammonia so that pH goes up. The color of agar media then changes to indigo blue, from yellowish green. The media is called CGB agar and is now commercially available.

I made the media and said, "This is the media with which you will be able to tell the difference between A/D and B/C without going through serotyping, that involves rabbit injecting, antigen making etc.," because at that time there was no commercially available antigen-antibody testing kit. Serotype testing was a really big deal because you have to produce antibody and then you have to absorb the antibody with yeast cells of different serotypes so that the antibody would specifically react with A only, B only, and so on. When I made this media, based on the biochemical differences between the two species, an Australian scientist named David Ellis [Dr. David Ellis] used this media to isolate strains of *C. gattii*. He swabbed barks, dead leaves, etc., of *Eucalyptus camaldulensis* trees and isolated *C. gattii* strains for the first time from nature. First of all, he was using the bird seed agar (Staib agar) to isolate melanin forming cryptococci. For any yeast which produced melanin and grew at 37 degrees C., he asked, "What serotype of *Cryptococcus* is it?" He was using my media. Lo and behold, serotypes B and C came only from the trees, and the tree debris, or soil contaminated with the tree debris, not from pigeon droppings. I think that my work opened up a new direction of studies on *Cryptococcus* ecology. Now more than 100 species of trees are known as the habitat for *Cryptococcus gatti* serotypes B and C.

Harden: You also contributed in a major way to the molecular genetic control of the fungal mating system. I am thinking of Joe Heitman [Dr. Jospeh Heitman] and his trainees at Duke. You did the early mapping?

Kwon-Chung: Yes, Joe Heitman was highly trained in molecular biology, molecular genetics in the *Saccharomyces* field, signaling pathways and the signaling pathway that leads to mating. He was highly equipped and trained for some serious work on the cryptococcal mating system. In the early 2000s, he moved into the cryptococcal field. I mean that's like a nugget, picking nuggets everywhere. He expanded greatly the details of this mating system and the signaling pathways involved in mating and regulation. Tremendous. He really energized the field.

Harden: I was struck by your combination of basic research and collaborative clinical research papers, especially in seeking therapies for fungal infections. Would you talk about this in the context of being here at NIH and the ability to work with collaborators like Jack Bennett, John Gallin [Dr. John Gallin], Harry Malech [Dr. Harry L. Malech], and Steve Holland [Dr. Steven M. Holland].

Kwon-Chung: John Gallin, Harry Malech, and Steve Holland are interested in patients with a primary immune deficiency like CGD, chronic granulomatous disease. This is a genetically inherited immune deficiency that affects the NADPH oxidase system. These patients are very vulnerable to fungal disease, especially with *Aspergillus*. If a new species of *Aspergillus* or very unusual species of *Aspergillus* gets into these patients, they contacted me, and this is how I got involved more and more in the clinical setting, knowing that fungal disease can be very serious in patients with primary immune deficiency.

Harden: This was perhaps easier to do here than it would have been at the university?

Kwon-Chung: Oh yes, yes, yes, because the clinical lab is right at the second floor. They isolate fungal strains from patient. Now they use a molecular approach PCR the fungal ITS genes and sequence or use the MALDI-TOF system and figure out what species they have without knowing what the fungi look like or how they behave. In earlier days up to early 2000, you had to know about fungal growth characteristics and their morphology by examining them under a microscope. I was a reference person when an unusual fungal pathogen was isolated. Then I usually got to see what the fungus looked like in histopathology of infected tissue. When they had not isolated a fungus, they would ask me to look at the biopsy section and ask, "What do you think this fungus is?" I might not be able to name the species, but I could often get to the genus, and that would help for treatment and understanding the disease.

Harden: You sponsored many Fogarty International Center visitors and trained many people during your career who have gone on to prestigious careers themselves. Would you comment a bit on your philosophy about how it's best to foster young scientists?

Kwon-Chung: I didn't actively search and try to recruit. Young scientists would write to me and say, "Can I come?" After reviewing their credentials, I would ask, "What do you want to work on? What do you want to do? Are you interested in *Cryptococcus* or interested in *Aspergillus* or interested in *Candida*?" I used to work with all three organisms until the late '90s. Then, if the scientist had very good recommendation letters, and if I had the resources--in other words, desk space or lab space--I would usually accept them. That's why I had so many international visiting fellows. They came from all over, especially fellows from Israel. They were very productive and I had very good relationships with them even after they went back. They all were successful in their career, and one them just retired two years ago from Hebrew University. He was one of the early post-docs who worked on melanin and the biochemistry of melanin, Dr. Itzhack Polacheck.

I don't micromanage. That is one thing I can say. I always have a one-to-one dialogue with fellows in addition to our once-a-week lab meeting. They are free to walk in and ask questions or tell me what they're doing. I think that micromanaging is not my character. I can say, "Let's work with this organism and look into this direction," but I don't tell them every step of what to do. I should say, I have been very fortunate to have very qualified fellows, and then after they went back to their country, they all did very well.

Harden: One last question in this theme. As a woman in science, you must have experienced from time to time, shall we say, feeling like you were second class or whatever, and yet you came through, triumphed. You had supporters, mentors. You have any comments on that or comments for young women who are going into science now?

Kwon-Chung: They are much better off than when I was their age. I became a section chief after I was, what, sixty-one or sixty-two years old. My son said, "Mom, you are the oldest rookie at NIH." I still remember that. I don't know how much of femaleness had anything to do with taking that long. Nowadays, it is a different world. Then, I was the only female PI [principal investigator] in the Laboratory of Clinical Investigation, NIAID, and it was not very comfortable. I was only PhD, the only female, and the only minority race, so I was a triple minority. Everyone else was a physician, and I was the only PhD. You can imagine, it wasn't easy, but I think that perseverance is even more important than IQ in a scientific career. When you really want something so badly and you want to give everything to make it and even if the environment is unfriendly or not comfortable, or you are not treated as an equal, you can make it.

Harden: That's the end of my questions. Is there anything else you'd like to add?

Kwon-Chung: I'd like to add that in 1982, I was awarded with both the International Society of Human and Animal Mycology Award and the Lucille George's Award given by the International Society of Human and Animal Mycology (ISHAM). At that time, I was probably in my early forties, and I had made an agreement with a publisher to write a medical mycology reference book within three years. I started it in 1979, and I was under great pressure because it was not a multi-author kind of book. It was Jack Bennet and myself alone as co-authors. I think I gave you the picture of us when the book was finally published in 1992 (K. June Kwon-Chung and John Eugene Bennett, *Medical Mycology*. Philadelphia: Lea & Febiger, 1992).

Harden: Yes.

Kwon-Chung: You can imagine how it is to be working full time, and when you go

home, you have three kids, and everybody wants your attention. After dinner I would clear the table and then work until 2:00 am. The stress was too much, and I developed really very bad inflammatory lesions in my stomach and so on, and I began losing weight. It was really very difficult.

One day, I got a letter that said, "You are the winner of International Society of Human and Animal Mycology Award and Lucille George's Award, which has been just established. You will come to New Zealand to get these awards." Well New Zealand is not next door, and at that time, foreign travel was very much restricted because of a budget squeeze. We had to get special approval for the foreign travel. I showed that letter to our intramural director at that time, Ken Sell, and he said, "This big award, so of course we have to support your travel." The awarding society was providing the airfare anyway, as well as housing, food, etc. I went to New Zealand, and there I realized how much stress I was carrying on my shoulders, writing the book, working full time and being a full-time mom plus interacting with all these colleagues. In New Zealand, the university town of Palmerston, there's nothing you can do in the evening. The university setting is in the middle of farmland. What do you do in the evening after meetings? Everybody's drinking and socializing, but because of my stomach condition, I couldn't even touch the alcohol. So I rested.

That award really gave me a boost, however, toward finishing this book because everybody was expecting me to finish it. Even if it took another ten years, I determined that I was going to finish it. The book finally came out in 1992, and I'm so glad that it took so long because if it had come out earlier, AIDS was not at its peak and there would have been no appreciation of cryptococcosis as a defining AIDS disease.

Harden: That's right.

Kwon-Chung: Cryptococcosis and mycotic disease was always considered as less important than some other diseases until AIDS appeared, and then cryptococcosis started making the news and the *Candida* thrush, and aspergillosis. The book's first printing was sold out within six months. Why? Because every clinical lab had to know about the association of fungal disease with this immune deficiency—we used to pronounce all four letters of A-I-D-S and now call the disease HIV/AIDS. I think that the award that I received in 1982 gave me a new kind of impetus to finish this book. When I look back, I'm glad it came out in 1992 instead of 1979.

Harden: Thank you so much for an excellent interview, Dr. Kwon-Chung.

End of Interview