An Interview with M. Gerald Neuffer

M. Gerald “Gerry” Neuffer received his bachelor's degree in agronomy from the University of Idaho in 1947 and the doctorate degree in field crops from the University of Missouri in 1952 under the mentorship of Lewis J. Stadler. Following a short postdoctoral position at the University of Missouri, under both Stadler and John Laughnan, Neuffer was appointed assistant professor in the Department of Field Crops at the University of Missouri in 1951, and after Stadler’s death in 1954, in 1955 assumed the university position previously held by Stadler. He was tenured and promoted to associate professor in 1956 and full professor in 1966. He chaired the Department of Genetics from 1967 to 1969. He retired from the Department of Agronomy (now, Division of Plant Sciences) in 1992, and he currently holds the title of professor emeritus in the Division of Plant Sciences.

Neuffer has had a tremendous influence on the history of maize genetics over the last half century. His early research has contributed to our understanding of the compound nature of the *R1* locus, the characteristics of the compound *A1* locus and its response to the *Dt* transposon system, the discovery of the aleurone and plant, color factor *bz2*, tetrasporic embryo sac development, the paucity of auxotrophs (Sheridan and Chang, 1994). He is credited for developing, with his long-term colleague Edward H. Coe, the paraffin oil method for treating corn pollen with ethyl methanesulfonate (EMS) and nitrosoguanidine (NG) as well as for the use of chromosome breaking Ds method to study chromosome structure and gene function. He is perhaps most well known for isolating and cataloguing thousands of mutations in maize and his generous distribution of his collection to researchers across the world. His willingness to share his mutant collection so widely led to a number of additional contributions, including the discovery and analysis of defective kernel (dek) mutants and endosperm-embryo interaction, with William F. Sheridan; disease lesion mimics and their relationship to disease resistance, senescence, cell-cell signaling and aging, with David A. Hoisington, Virginia Walbot, and Gurmukh S. Johal; and duplicate factors for orange pericarp and the auxin pathway, with Allan Wright.

Neuffer is author or co-author of numerous refereed journal articles as well as author and/or editor of two books. Among his publications is *Mutants of Maize*, an authoritative reference on mutant phenotypes in maize co-authored with Loring Jones and Marcus Zuber in 1968 and then updated and expanded in 1997 with Coe and Susan Wessler. Neuffer is also highly regarded for his ongoing service to the larger maize community and his leadership and central role in building the maize genetics research group at Missouri.

On July 3, 2009, James Birchler, Curators' Professor in the Division of Biological Sciences at MU, sat down to interview M. Gerald Neuffer. The interview took place in room 219 of Curtis Hall, the building on the Columbia campus of the University of Missouri that has been Neuffer's academic home since 1947. Following are excerpts from Birchler's interview with Neuffer. Some of the questions and answers have been edited for conciseness and clarity, extraneous material omitted, and footnotes added for elaboration or clarification.

Early Years and Influences

BIRCHLER: I recently read this quote by Craig Venter: "Like so many people who have succeeded in life, I have had some great teachers who encouraged and inspired me, taking a real interest in my education." Who might you attribute as an inspiration to you to get into genetics in your early days? How did you intend to go into this career?

NEUFFER:[1] Well, I didn't intend to go into corn genetics initially. But Herman K. Schultz, a student of Dr. Hayes and a wheat breeder in agronomy at the University of Idaho, taught my genetics class. He said that I ought to do something in genetics rather than plant breeding and recommended I go to Missouri and study under Lewis J. Stadler.[2] At that time, there were four people that he would recommend: Ernest Brown Babcock at the University of California Berkeley; Ernest W. Lindstrom at Iowa State University; Herbert K. Hayes at the University of Minnesota; and Stadler at Missouri; and Cornell to pursue Emerson's earlier work I applied to all of them. At that particular time, I was married and had two children and didn't have any money. One offered me an assistantship, and one offered me a place to live, and the rest didn't offer me anything except an invitation to come. Stadler offered me an assistantship, and I took that. I was glad I did because Babcock was at the end of his career, and Lindstrom died before I finished up. I certainly made the right choice.

Stadler has been the most influential in my professional life. He was what I needed. I was an innocent farm boy who didn't have any really deep training in biology, just a lot of good experience in agriculture. I was a generalist, and he took a hold of me and taught me lots of things. He was the kind of person who brought up new ideas every day. He'd go home at night and think all night long about a new idea. The next day, he'd come in and say that we should

[1] For more on Neuffer's early years, see (Sheridan and Chang, 1994).

[2] For a portrait of L. J. Stadler, see (Rhoades, 1957).

work on it. And I'd work on it all day. And the next morning, he'd come in and have another new idea before I had finished the first one. I asked him once if we could finish just one idea, but we did not often get to do that. Even now, I look back at his old black pocketbooks where he'd write down his new ideas. Later in my career, I'd think I'd come up with a new idea, and I'd look in those books, and he had already thought about it.

BIRCHLER: What year did you come to Missouri?

NEUFFER: 1947.

BIRCHLER: When you came to Missouri, did Stadler already know he had leukemia?

NEUFFER: Yes. A month or so after I came here, he came in smiling one morning and said, "I just received word that I have remission from leukemia, Hodgkin's disease." He was doing very well recovering. And even though he was quite ill, his brain was really sharp. I think he was very happy that day….His health was good for a while, but then he had some limitations, and he started leaning on me to handle things. He'd turn things over to me when he was not feeling well. I would do the job and report back continually. I got the Genetics Farm started and kept things going as he had planned them.

BIRCHLER: What were some of your more interesting or fun interactions with Stadler when you were a graduate student?

NEUFFER: The thing I need to tell you about is I came here not really prepared. I had a good reputation and undergrad training, but I was not really the best student. I got a C in genetics and a B in organic chemistry, just from poor study habits. But I had a really good basic understanding of farming and of living things, and I really had a good instinct for how things worked. Stadler understood that I needed training in scholarly scientific procedures and tried to bring me in contact with people that were precise in their thinking. One day during a conversation, he called the bookstore and asked them to send me a copy of the book *The Logic of Modern Physics* by Bridgman. He said that Mendel took the operational approach and so should we. Stadler was a person who didn't direct you in detail. He just tried to give you ideas and let you carry them out. He started me out on a thesis project to determine the behavior of genes and chromosomes during microsporogenesis using X-ray induced deficiencies to mark the various stages of development. In this way, I was able to learn precisely the position and activity of a particular gene, chromatid, or chromosome during male germ cell development. This became

very useful to me during my later efforts in mutagenesis, even though I never completely finished the project. I used the techniques in later experiments to locate genes by X-ray induced losses in male germ cells and to analyze the pachytene configuration for *Bz2* and *Dt2* losses.
BIRCHLER: In your graduate studies, was Stadler already working in Curtis Hall?

NEUFFER: Yes. Curtis Hall had just been going a few years. It was built in 1939, and I came in 1947. Emil Heitz[3] of Drosophila salivary gland chromosome fame was here, and we shared an office-lab room together up in 301 [Curtis Hall]. We had Alexander Cyril Fabergé[4] in Botany doing irradiation, and Jesse Singleton [also] in Botany, who was a replacement for Barbara McClintock, doing cytogenetics. He was really quite good. And we had some really good people in Zoology, A. B. Griffin doing *Drosophila* genetics and Dan Mazia doing cellular physiology.

BIRCHLER: Who instructed you on cytology?

NEUFFER: Joe O'Mara was supposed to do this, but Joe had other things to do. So Jesse Singleton took over my training. Stadler just kept after me and told me what I should be reading. He just kept talking to me about what was needed to solve the problems. I chose the things that looked important, and that's the way I picked it up.

BIRCHLER: Did Stadler ever do any cytology?

NEUFFER: Not really, but he seemed to know all about it. He looked into my microscope and said, "Yes, this looks good" and "Oh, did you see that?" McClintock had been here just before [I came], and she left a lot of tradition, and a lot of people around here were thinking along those lines. But, actually, nobody really did corn cytogenetics but me. I used the understanding of meiosis in a lot of things I did later, and it was a nice thing to have that experience

On Becoming a "Half Stadler" at MU

BIRCHLER: How did you transition into your faculty position at the University of Missouri from being a graduate student here?

---

[3] Emil Heitz was a German-born cytologist who worked with both animal and plant tissues. L. J. Stadler invited him to come to the University of Missouri as a visiting professor. For more information, see (Kass, 2005, pps. 13-14).

[4] Fabergé was at the University of Missouri from 1974 to 1955. For a portrait of Fabergé, see http://www.utexas.edu/faculty/council/2000-2001/memorials/Faberge/faberge.html

NEUFFER: I was the last student that really had serious involvement with Stadler's maize genetics project. I was an average graduate student. When Stadler got really ill, he was down in Barnes Hospital (in St. Louis), and he called Dean John Longwell down there, and they talked. I didn't know until some time later what they talked about. But one thing that I did learn is that Stadler made Longwell promise that he would take care of his corn genetics project. Longwell was the old-style kind of scientist/administrator: when he made a promise, it was a promise. And essentially he kept that promise as long as he was dean and then he transmitted that promise to his successors. So [Stadler's] project was protected, absolutely. Stadler was hired by the USDA and the University jointly. When he passed away, they wanted a successor. I had already received my doctorate and was postdoc at the time. [Stadler] wanted John Laughnan [to replace him], so the dean hired him. John Laughnan came here for a year, but then Illinois wanted him back as the department chairman of botany. Illinois had bigger guns, and they hired John back….When he went back, the question came up, who would take his place? At that point, the USDA was looking too, and they that they would hire somebody and the University would hire somebody. So when the [USDA] position came open, I applied and Ed [Coe] applied. Ed came and interviewed here, and he got the job. I was disappointed, but it actually turned out better for both of us.

BIRCHLER: Did you know Ed?

NEUFFER: I didn't know him before. But, I remember when he came, he did a good job at the interview, and they were impressed, and the USDA hired him right on the spot. Well, then Dean Longwell called me and said, "We'd like you to fill the University position." And then a very interesting thing happened: I had an offer to go to Iowa State. I went up and interviewed for the position that I think Pete Peterson [eventually] took. On the committee that interviewed me was the prime statistician at Iowa State. He said, "Neuffer, I read your thesis. You didn't have a thing on statistics in there." And I thought, "Oh, man, here I go, down the tube." But I was ready for it, and I said, "I planned it that way. With mutation and cytogenetic research, we're not talking about statistics. We're talking about things that happen or don't happen. I set all of my experiments up for 'yes' or 'no' answers." That satisfied him, and they offered me the job for $6000 a year. Dean Longwell said, "We can meet that." I said, "Okay, I'll stay here." Alex Faberge was here, and Faberge was getting $5600 a year. He was in the Botany Department. I had taken classes with him. Faberge found out that I got $6,000 a year. When he heard that, he was furious. So he sat down and wrote a letter of resignation to [Dean Longwell] and [also] sent that letter to everybody in the Genetics Society. [In it, he said] that he only gave me As in his

class because I was Stadler's student, that I was the poorest student he ever had and I went to sleep in all his classes, that I was a really bad choice, and he was therefore resigning in protest. I heard about that, and I went to see Dean Longwell. Dean Longwell said, "Well, Gerry, we accepted his resignation. Does that answer your question?" But my career was *shot* when that letter went out. I could never have gotten a job anywhere with that kind of letter on me. But then John Laughnan went around to the members of the Genetics Society at the annual meeting and put matters straight. I'm grateful to John for that, and I'm grateful to Dean Longwell.

So Ed was hired to fill the Stadler position with USDA, and I was hired to fill the Stadler position with the University. We often laughed and said that we were each "a half Stadler" and it took the both of us to do the job. There was a lot of truth in this. We and our respective organizations were exceptionally complementary. For example, it was Ed's discovery of the use of paraffin oil that started me on my way to successful chemical mutagenesis. I am still amazed at the collective wisdom and foresight of the USDA and University officers that made this all happen. It certainly could not happen under current conditions.

When I took over Stadler's project, I didn't realize what I had gotten a hold of. I had a budget of $16,000. I essentially had control over Curtis Hall. I had control of the Genetics Farm. I had two postdoctoral fellowships, four graduate student assistantships, a secretary, a technician, a field foreman, and the best maize genetics stocks in the world. And that $16,000 was with no strings attached. I could use it essentially anyway I wanted. All I had to do was make an annual report. And not only that, I was protected. So, you see, if I hadn't succeeded, it would have been tragedy. It would have been a shame to fail with those kind of resources. I didn't even have to apply for tenure. I got a call saying, "Gerry, we just turned your name in for tenure. You'll get Associate Professor and a good raise." For a long time, I thought that's the way things went in the world. It's not true.

BIRCHLER: [laughs] Whatever works.

NEUFFER: I've been grateful. I'm grateful for a lot of things. And it's people like Longwell and Stadler and Sears. Sears was a wonderful person to work with because he was so unassuming. He taught me that you don't go out and blow your horn and propagandize trying to convince people you're good. You just go out and do your work and occasionally publish…. You're a public servant. Everything you do is public property. So you can't go out and patent it. You're not worried about whether somebody is going to scoop you or not. If you're work is good, you will share it and that will benefit all of your colleagues. The attitude of keeping what

you know to yourself so nobody will scoop you, just never crossed their minds. And Ed is a good person to be with in that same regard. Ed's been a marvelous colleague, and we've done all kinds of things together. It's been a real good experience.

The Origins of the Chemical Mutagenesis Work

[O]ne day while working with *a1Dt1* (Rhoades), I had an ear of colorless kernels, each with a few scattered purple dots. I saw one kernel with hundreds of dots. I wanted to know what had happened, so I planted it and began investigating its progeny. I just decided to pursue it further. And later on, I guess, Stadler got the idea that I was going to do this and did not object, so I made it my project. Of course, it became a question then whether *a-m Dt* was similar to McClintock's *Ac/Ds*. For a while, I thought they were entirely different systems and that I was doing an independent parallel analysis. But it soon became apparent that it was just a matter of the particular behavior of the transposon in the *dotted* system as compared to the same thing in the *Ac/Ds* system. It was a part of that work that led me to chemical mutagenesis, which was equally exciting.

BIRCHLER: When you started in chemical mutagenesis were you trying to mimic *Dotted*? What was your inspiration?

NEUFFER: Yes, I guess I was trying to mimic *Dt* in a way, but I was also trying to do something operational that would specifically replace *Dt* and the difference would tell us something about the characteristics of the *a-m Dt* transposon system. A mutable *A1* allele (*a-m*) had occurred when a responding component of the *Dt* system had moved into the *A1* locus. The hypothesis was that the *Dt* receptor moved into the *A1* gene and stopped *A1* function, producing a colorless (not purple) kernel with purple colored dots (reversion to *A* function due to removal of the *Dt* responding suppressor) as long as the *Dt* activator is present. If by segregation the activator *Dt* is separated from the suppressed functional *A* allele, the suppression remains in force, producing a completely colorless kernel with no revertant dots. This produces an amazingly stable *a-m* allele in the absence of *Dt*.

The plan was to try to remove the suppressor transposon unit, in the absence of the *Dt* activator unit, using known chromosome breaking agents (ionizing radiation) and mutagens (UV) and the chemical mutagens EMS and NG. If such an event were to happen from pollen treatment, the expected result would be a single purple kernel in a large population of colorless kernels with no dots (assuming single strand chromosomes in the pollen grain) or partial colored kernel sector if the chromosome was double at time of treatment. The results were surprising. Not a single

example of a full purple kernel or a partial sector or a single colored dot was found in huge populations. Instead, we found several sectors of dots from all the treatments. At first, we thought that the treatments had caused the removal of the transposon directly in bursts, but we concluded that the treatments had produced a new *Dt* (activating agent) that subsequently continued to remove the suppressor in subsequent cell generations. This really was not surprising because McClintock showed that transposon systems arise as a result of chromosome breakage. Our subsequent analysis of the progenies of these treatments revealed huge numbers of new mutants of many genes of all kinds and led me to follow chemical mutagenesis as a project instead of transposon analysis.

BIRCHLER: Was it Ed Coe who first realized that pollen was viable in paraffin oil?

NEUFFER: Yes, Ed discovered the paraffin oil technique of making pollinations.

BIRCHLER: Do you know what his inspiration was for putting pollen in paraffin oil?

NEUFFER: He wanted to use paraffin oil as a carrier for chemical screens to select for genetic traits in corn pollen. The paraffin oil preserved and did not kill the pollen. But then the problem became one of finding out how to use some mutagenic agent in paraffin oil. I tried everything that I could get my hands on. I tried nitrosoguanidine, EMS, and a number of other things. Nitrosoguanidine was effective but was deactivated by sunlight, and I couldn't get it stabilized. EMS appeared to be very stable, so it was just a matter of finding out the right dose and the conditions for treatment. I spent a lot of time doing that. I had a graduate student, named Gyula Ficsor, who spent his whole graduate time trying to figure out ways to do this. You really have to get the right stage of microsporogenesis: if you treat too early you get somatic sectors with multiple gametes carrying copies of the same mutation; if you treat later, you get only samples of the mutations actually produced. We also used to take corn plants to the medical school -- in collaboration with Dr. Henry McQuade -- and treat them with radiation (tritiated thymidine). We tried seed treatment, too. Seed treatment didn't work, because when you treat the seed, you treat all cells in a multicellular embryo, the progeny of which has mutant sectors, and then you get multiple copies of the same mutation. That is not very rewarding. But pollen is perfect because you have one cell, with one strand or two strands, then you know what you have, and there is no ambiguity.

I tried to report the results of my experiments with *a-m Dt* in a paper that I sent to [Marcus] Rhoades, to put it in the *Proceedings of the National Academy of Science*. He sent it back and said, it unacceptable. I know now that he sent it to McClintock, and she said it wouldn't go.

BIRCHLER: Was it ever published?

NEUFFER: Just pieces of it. The whole paper was never published as such.

BIRCHLER: So, you took the stable *a1* allelle -- that's a *Dotted* responder -- and UV irradiated it, and you got reversion. Is that what happened?

NEUFFER: Yes. I did get sectors of dots (reversions), but no I did NOT get single dots or colored sectors, which would have been the case if the treatment had directly produced the reversion.

BIRCHLER: You sent that to Rhoades to communicate to *PNAS*, and he had sent it to McClintock, and she recommended it not be published.

NEUFFER: She was right. And I know why, now. I was saying we have something that is absolutely stable under one set of circumstances and mutable in others. The only thing I could think of to explain that [phenomenon] is that it is a hole, or empty space, an absence. You can't take a hole and make it into something. But you can take something that's there and change it into something else. I shouldn't have said that in the paper, because I was just speculating. I guess McClintock didn't like that. So [Rhoades] sent it back.

BIRCHLER: We probably now know that you mutated the transposable element.

NEUFFER: Yes, the activator component Dt was changed as a consequence of chromosome breakage, but the receptor transposon was not changed. With X-rays I got a sector of dots. I did not get a single half seed sector or a whole seed or a single dot. I got sectors of dots. But with X-rays, with UV, and with EMS, all of them I got sectors of dots. So my conclusion was that I was not kicking something off but that a new *dotted* was being made, and that's what I said in the paper. But I didn't know what it was because at that time I didn't understand what DNA was all about.

BIRCHLER: Yes, well, nobody understood.

NEUFFER: So I'm a bit sad that I couldn't communicate better. It happened that some years later McClintock gave a talk in Atlanta. I didn't go, but Ed came back and he said that McClintock got up and said, "You know, Neuffer up in Missouri has some real good ideas on transposable elements and you should pay attention to that." That's as close as she came to apologizing for killing my paper.

The Rise of the Mutants of Maize

BIRCHLER: Did you ever imagine that EMS would be as effective as it turned out to be?

NEUFFER: No, not really. Actually, I worked real hard to get the system to the point where I got mutations. I had lots of false starts, and it was so hard to get something that really caused single gene changes. I had to take the pollen and grow it on agar and use anything that had any effect at all on pollen germination. If you'd see an effect in the pollen, it was too much. It would kill it. I did lots of these kinds of tests. I finally got to the point where it worked. The first few experiments that I did were the best because I was careful. I spent a lot of time getting the conditions just right. Then I suddenly discovered that I had more mutations in my laboratory than the rest of the world.

BIRCHLER: What was your first indication it was working?

NEUFFER: Well, I grew out the M1 from mutated pollen. I found a number of different dominant mutants. The most frequent was oil yellow (Oy). I used that as a measure of successful treatment. If I could see oil yellow, I knew it was a good treatment. If I had been working with A632, I couldn't have used that because oil yellow is suppressed by A632.

BIRCHLER: What was your reaction when you realized that?

NEUFFER: My reaction was, What do I do with all these mutants? I talked with some friends who were working in *Drosophila* -- Jerry Braver and Mel Green -- and I said, "What should I do with these mutations?" They said, "Well, we have mutations flying around our lab all the time, they are so common."

BIRCHLER: [laughs] So to speak. Literally.

NEUFFER: Right. They weren't impressed. I think that mutant variation is much more appreciated by those working with crop plants and farm animals. I decided this was pretty exciting, that it was something worthwhile. I thought I could go get some money to do it, and I planned to write a grant proposal for it. I talked to Ed about it. He looked over the proposal and said, "Well, Gerry, people out there nowadays are not impressed with lots of mutations. I don't think you're going to get much for that." But, I went ahead and did it anyway, and I got my grant. I got all the money I asked for the first time. That was through the USDA.

BIRCHLER: So with your initial studies with EMS, you were looking for *Dotted,* and then it just rained mutations. You had more mutations than anybody else and since, I believe. What did you do with them?

NEUFFER: Yes. I wanted to cause changes in the mutable *a1-m* allele in the *a1-m Dt* transposon system. This allele of this system is completely stable in the absence of *Dotted*. I wanted to find a mutagenic agent that would change that stable condition, thinking that it would be a positive mutation change. But instead I found what appeared to be good single-gene changes at other loci. I have a picture of my cornfield of 8000 M1 plants. The mutation frequency in that field was something like one mutation per locus, per thousand of pollen grains treated. In that field, statistically, I probably had a majority of the mutations that exist. And I had to decide what to do with them.

I decided I couldn't possibly deal with them all alone. I could pick out one or two of the most interesting and make a career analyzing them. It was popular in corn genetics research to pick something, some locus, and work with it that way. But I decided that I wasn't going to do that. I was just going to push the whole collection and make them available for colleagues to use. I was going to watch over them and make sure that they were used properly. I sent out a letter to everybody saying, "I have in my hands M2 materials. If you come to my lab and get a 20-seed sample from 3,000 or so, you will have a 95% chance of getting almost any mutant you want. Just come and get them." And a lot of people came and did this. By my doing this, I got a lot of good will among my colleagues. I could have been possessive of them, but I wasn't.

Don Miles was looking for some mutant controlling electron transport across the chloroplast membrane. I said, "Well, Don, I'm running M2s in the greenhouse this winter. If you just come over to my greenhouse once a week with your UV lamp and a red filter and look at my M2 seedling materials, you'll find lots of mutants." He thought he'd find one or two loci in there. Well, he found 54 cases that amounted to 19 loci. I wish that I had pushed a lot of other people to

view those M2s. Bill Sheridan was at Missouri at the time. He said he was interested in defective kernels and embryo development. We took a bunch over to him. And so that was the way I did it.

Unfortunately, when I retired in 1992 and my replacement left Missouri, most of the material was thrown away. I'm not very happy about that because I'd still like to be able to say, "Come here and get an M2 and get whatever mutants you want."

BIRCHLER: So the defective kernel collection probably still exists?

NEUFFER: Yes. And I had the lesion mimics. Seed samples of most of the mutants that I was following have been sent to the Maize Genetics Coop and descriptions and photos of all of them are posted at MaizeGDB. Since retiring, I've been trying to re-establish a big M2 collection. I've not been entirely successful. If I had a career left to do, I'd do precisely what I have done, namely "mutagenesis on call."

BIRCHLER: So you have sent off your mutant collection to various people. Do you think most of them have done stuff with them?

NEUFFER: Actually I invited them to come to our laboratory and collect statistically significant (3,000 20-seed) samples of our best M2 to use in their search. Many important research projects were based on these collections from the EMS M2. I've gained a great deal from these invitations since my retirement. Friends and colleagues have gone to great lengths to support my efforts to bring the EMS collection to the attention of all those who might profit by having so many mutants.

Disease Lesion Mimics

BIRCHLER: What attracted your attention to the disease lesion mimic mutants?

NEUFFER: Well, I had the oil yellow mutants as a signal and as a measure of the effectiveness of each treatment. I also found in this material that I had dominant lesion mutants. Oil yellow has a frequency of about 1 in 1,000; of course, that is the recessive rate, even though it's a dominant mutant. I found the lesion mutants at about the same total frequency, but they were almost all different loci and therefore had a low individual frequency.

BIRCHLER: What were the first lesion mimics that you saw? Were they really dramatic phenotypes?

NEUFFER: One was really pretty dramatic. It had nice big lesions all over the leaves and was highly responsive to temperature. I had a long period of time in which I had to prove that there was no disease organism present. I had to grow them under sterile conditions in the growth chamber, take pollen out in the field, and pollinate something else. I finally proved it. I went to a lot of trouble with those first few. But there are many differences among them. There are many loci. At the last count, there must be over 100 cases. We found eleven of them with no duplicate loci. My friend, Albert Romano, a mathematician, says, when you get eleven cases with no duplicates, you have over 200 variables. That was amazing to me. I feel that biology cannot afford to waste energy on things that are nonessential. If there are 200 loci, it must be very important, and it turned out that it was. We've had a hard time getting people to take them seriously. The plant pathologists don't want to talk to us about these things, and the geneticists aren't impressed. There are recessives as well. One of the major reasons for my continuation of research after retiring is to make sure that the lesion mutants are not lost, only to be rediscovered at some future time. The ones I have been working on are all dominants.

BIRCHLER: Have you ever examined the recessive disease mimics?

NEUFFER: I have not followed the recessives, but they are very important. I found that EMS produced both dominant and recessive lesion mutants but the transposon systems, like *Ac-Ds*, appear to produce only recessives.

BIRCHLER: Does UV irradiation cause any dominant mutations?

NEUFFER: I'm not sure. My experiments were not properly designed for that. Even though possible cases were quite frequent, there are lots of things that look like dominant mutants. I look through an M1 field, and I'll find 500 that look like dominant mutants, but only 25 of them are actually dominant mutants. It turns out herbicide damage, insect bites, and smut produce nice looking lesions. The frequency of actual good cases from UV was not significantly above the control.

BIRCHLER: Do you have speculation on why EMS caused the disease lesion mimics, whereas X-rays and transposons do not?

NEUFFER: Well, X-ray induced changes involve gross changes that are not generally transmitted. I don't know why transposons don't do that. I think they ought to.

BIRCHLER: So are the disease lesion mimics dominant negative? Have you ever tested whether or not that would be a possibility or whether they're a loss of function or antimorphic?

NEUFFER: No, they are not negative or loss of function.

Current Studies

BIRCHLER: What are you working on now?

NEUFFER: I saw so many recessive mutants. You see the same ones over and over again. And, on occasion, you'll find a recessive that is unique. I thought, the dominant mutants are rare, and that means I could still look for them in the treatments and find new ones all the time. Every time I do a mutagenesis, I find some new ones. So I decided it would be easy because I have colleagues doing EMS treatments, and some of them will let me look at their M1s. The corn breeders don't care much about dominant mutants unless they have some immediate application. I thought I could do that as a retirement project. I have about 200 new dominant mutants now. And I'm finding lots of exciting things.

One of the most exciting is this business about having whole kernel, whole plant mutants and also half plant, half kernel chimeras from the same pollen treatment. Evidently, the chromosomes are effectively double, and sometimes not, in the mature pollen grain. Sometimes EMS gets both of them when they are functionally one or can only get one when they are functionally double, at about the same frequency. These chimeras become a lot more interesting when you recognize that you can get transmission from them, even of dominant lethal mutants. If the mutant is lethal, you can't get progeny from, for example, an albino plant, but you can have an albino chimera and get progenies from it, if you were wise enough to make a lot of pollinations from the chimeric plant. At first, I didn't realize that these were so valuable, but then I suddenly realized that they were the best ones. If you see a whole plant lethal case you cannot get a pollination from that outcross, and you lose it. If you see a chimera, you see tissue that's mutant and tissue that's normal on the same plant, side by side; you can see and compare it in the same background, and you know just exactly what they look like. If you don't get transmission, it can be either of two things: either the sector is not included in the tassel, which is pretty rare, or it's something that, when outcrossed with something else, doesn't express itself as a dominant. I now

have quite a number of dominant lethals that aren't much good, except as you outcross them you get them in a different genotype. This brings up something that's important about genetics: if you put a gene that's lethal in one genotype into another genotype, it may be viable enough to get progeny. That's really true. If when you make the outcross, you get the heterozygote, then it's a hybrid. If you grow and self it, then you can get variants that have enough modifiers to overcome its lethality.

BIRCHLER: Did these chimeras come directly from an EMS treatment and you found dominant in the mosaic versus a normal genotype?

NEUFFER: Yes, you find the same frequency of chimeras as of whole plant cases for each mutant. They're not all half/half, but typically, as shown by the frequency of mutant gamete transmission. I've had albino sectors. I've seen whole albino seedling dominants.

BIRCHLER: Right, so you have a dominant albino situation.

NEUFFER: Yes. So, I know if there's an albino mutant chimera situation, I can make an outcross, but I can only work with them as long as the outcross seed lasts. Some of them actually are conditional mutants that are near enough to normal that you can get progeny. I have a nice one that I'm working with now. It is a half plant: one half leaf is bright pale green and the other half is green. I outcrossed it and have progeny in the field right now. The plants are tiny, about 6 inches high, and pale green. But among them are also ones that are 14 inches high and are yellow green, but they die. I put some of them in the greenhouse with the high-intensity lights and ideal conditions. I got them to grow about 18 inches high, and they were actually just lighter green. But they fell over. I found that they didn't have any roots on them. I sent these off to a colleague who gave a talk about plant roots, and she was pretty happy about that.

BIRCHLER: What are you doing with Sarah Hake?

NEUFFER: Well, she's sponsoring my search for new dominant mutants. I have a lot of them, and I'm ready to turn them over to co-op now. I have pictures of them, which I plan to send along, with seed, to the maize stock center. I also have pictures and data, which I have sent to Maize GDB.

BIRCHLER: Do you have any striking mutants that no one seems to be working on?

NEUFFER: Yes, I do, and anyone can have them just for the asking. However, the most striking one I did not manage to save. I found it in one of my M2s being grown in Illinois. This was a beautiful thing. It's a dominant mutant. It looks like a leopard spot. I've seen it twice, so it must be a good mutant.

Important Questions or Problems in Maize Genetics

BIRCHLER: If you were giving advice to an up-and-coming maize geneticist, what areas are valuable things to focus on?

NEUFFER: I have a little different take on that. Soon after it was discovered that DNA was the genetic material, people working with viruses thought they had solutions to all the problems of inheritance. But they did not understand that these simple solutions were not the final answer. The paths that we see serve a purpose. They're valuable tools. But all these valuable tools are only just a part of the picture. You eventually have to get back and know the organism that you're working with, like McClintock said. You don't know where the next important discovery is going to come from.

BIRCHLER: Maize seems to have been denigrated as a model system over the years, but it manages to keep coming back. Why do you think that is?

NEUFFER: Interest keeps coming back to corn because it has special attributes that make it specially good for genetics research. So I guess I'd say to a corn genetics graduate student, take heart and it will be up sometime for you. But it's been marvelous for me. I started out in 1952. I've been able to have my whole career parallel from the discovery of DNA until sequencing. It's been marvelous to watch.

BIRCHLER: Gerry, it's been a pleasure. This has been a lot of fun. I've learned a lot of things from both you and Ed.

NEUFFER: It's been fun talking about it, fun thinking about it.

Acknowledgements

The editors of the *Maize Newsletter* would like to acknowledge Melody Kroll for organizing the interview and her assistance editing, footnoting, and transcribing the interview and writing the introduction.

References

Kass LB. 2005. Missouri compromise: tenure or freedom? New evidence clarifies why Barbara McClintock left academe. *Maize Genetics Cooperation Newsletter* 79:52.

Neuffer MG, Jones L, Zuber MS. *The Mutants of Maize*. Madison Wisc: Crop Science Society of America, 1968.

Neuffer MG, Coe EH, Wessler SR. *Mutants of Maize.* New York: Cold Spring Harbor Laboratory Press, 1997.

Rhoades, MM (1957). Lewis John Stadler (1896-1954) – A biographical memoir. *Biographical Memoirs* 30:329-347.

Sheridan WF, Chang MT. 1994. Myron G. NEUFFER: His contributions to maize genetics. *Maydica* 39(1):223-229.

Stadler,D (1997) Ultraviolet-induced mutation and the chemical nature of the gene. Genetics 145:863-865

Stadler, LJ and Uber, F (1942) Genetic effectsof ultra-violet radiation in maize. IV Comparison of monochromatic radiations. Genetics 27:84-118.