RUST RACE SITUATION AND OTHER SUNFLOWER DISEASES

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I was asked to talk about the sunflower rust race situation. I shall describe what I know, what I think I know, and then try to explain why I do not know more.

First of all, I would like to use the phrase "the classic pattern" for distinguishing rust races, and please understand that when I say races I really mean race groups. As far as I am concerned, we have not yet really defined races in sunflower rust. We don't have sufficiently fine differentials to make it possible to break rust collections down to races that are actually single biotypes. All we can distinguish are groups that behave alike. They may be broken down much finer as soon as we get additional differentials.

The pattern that I am talking about is in this table:

The "classic pattern" of reaction of sunflower differentials to rust race groups

Differentials	Rust race group 1	2	3	4
Universal suscept (S-37-388)	+	+	+	+
"88" source	-	+	-	+
"22" source	-	-	+	+
"41" source	-	-	-	+

S-37-388 is the female inbred Eric Putt used in his first hybrid sunflower. He has mentioned the 88 factor; it was the first source of resistance in a single plant, apparently an accidental cross, from material from Renner, Texas. The 22 factor had a somewhat different background, but the rust resistance came from the same place. The 41 factor was just a sister selection of the 22 but turned out on analysis with the rust populations available to have both factors, 88 and 22, which proved to be nonallelic, present in the same material.

We started out having nothing but sunflowers that were susceptible to all the rust that I threw at them. In 1949, the first resistant plant was found. In 1950, another resistant source turned up. In 1952, when we found the first rust resistant seedlings of more or less acceptable agronomic type which had been recorded in the greenhouse, we knew we had two groups of sunflowers, those which were susceptible, and those which were resistant. From there it was possible to differentiate further.

S-37-388 is susceptible to all the rust we have found so far. Source 88 is resistant to the first race group, susceptible to a second which I call race group 2, resistant to race group 3 and susceptible to 4. The 22 source is resistant to both 1 and 2, susceptible to 3 and susceptible to 4. Source 41 is resistant to race groups 1, 2 and 3 and susceptible to 4.

This is classic in the sense that it is what you can predict you will find when you have 2 distinct factors for resistance. Unfortunately, that is not the whole picture. I will start on the difficulties right away. First of all, we have trouble with our differentials. You have heard a fair amount from the plant breeders' and geneticists' point of view--self-incompatibility, self-sterility, etc. Well, there are other things operative here too. Eric Putt can't answer the problems I have raised on some of these things; therefore, I defy anyone to answer them because I think he is in a better position than anyone else. He produced a variety that is referred to as S-37-388RR, which has the 22 factor for resistance in it. I am very short of a good differential with this factor. A long time ago he gave me a selection from cross 69, 69-17, which was very good. But that selection is in short supply, and apparently very hard to produce.

A year or so before leaving Winnipeg some 4 years ago, I tested this S-37-388RR, and it behaved beautifully as the differential with the 22 factor. One of my graduate students working on the inheritance of pathogenicity in the rust pathogen was running short of this particular type of differential two years ago. I wrote Eric in desperation asking what he could supply, knowing that the 69 was hard to come by. He pointed out that S-37-388RR had the same factor. I naturally felt foolish because I had tested it very extensively at Winnipeg and it had been perfect. We got quite a generous supply of seed in 1962. Unfortunately, it no longer behaves as an acceptable differential with the 22 factor. It is not segregating out for susceptible material; it is more resistant than it has any right to be. Several other lines that I have worked with which were good differentials at one time are now more resistant than they have a right to be. Thus S-37-388RR, instead of being completely susceptible to races 3 and 4, occasionally tends to be moderately susceptible to 3 and is often moderately resistant to 4. This does not make sense, because 4 is supposed by definition to carry the factors for pathogenicity that attack both the 22 and the 88 sources singly or in combination. In the S-37-388RR we now find some apparent resistance to 4, and certainly more resistance to 4 than it has to 3. The same sort of thing is turning up but to a much lesser degree in the 88 source, the selection we call Cross 29. It is not resistant to race 4, but often it is not quite as susceptible to 2 as it should be.

The obvious thing for a pathologist to do when he encounters this sort of thing, especially when a graduate student reports it to him, is to chide him for mixing up the rust, not controlling conditions correctly, etc. And then I have had to be very contrite when the student drew my attention to subsequent inoculations with the same rust on material which gave the beautiful classic pattern in experiments parallel with those on the

aberrant S-37-388RR and on the repeatedly selfed Cross 29 which gave an aberrant pattern.

To sum up, then, we have trouble in keeping differentials which behave as they used to behave. If only we could get differential host material that would be as consistent as the lines Harold Flor has developed in his flax work, and the cereal breeders have produced in wheat and other crops, I for one would be very grateful. If any of you can suggest how you can consistently supply large quantities of material homogeneous for rust reaction phenotype, which can be used as differentials, you would do us a real favor.

One of the things that may enter here is a very definite sensitivity to environment on the part of certain host-rust combinations. I can't predict in advance which ones are going to be particularly sensitive, nor can I necessarily repeat the same results with the same combination. Very minor fluctuations in environment sometimes seem to be responsible for major differences in reaction which could tip the scale from the moderately resistant, moderately susceptible range to clearly resistant or susceptible. It may be a combination not only of light duration and temperature but of light intensity and also of light quality. I am very worried about this because even graduate students from the Near East who have more patience than many of us don't have an infinite quantity of time. And when they do an experiment repeatedly and each time it comes out a little bit differently and you are not quite sure what caused the difference, you feel very, very guilty about it.

To try and get over this guilt feeling, we have been doing preliminary work on a project which a student is going to carry as a Master's problem, to study specifically the effect of light intensity, day length, and temperature in various factorial setups within controlled environment facilities, on the development of rust on both resistant and susceptible hosts. I hope that by this time next year we will be able to predict a little more accurately just when we are going to get variation from expected results. At the present time, I can just report that it occurs. That is enough about the classic situation.

One of the things that I want to point out is that the deviations from the "classic" pattern occur in several different ways. First I must mention the distribution of the four "Canadian" races. All four were found originally in Manitoba material collected either on sunflowers growing in farm fields, in gardens, as volunteers in the wild, or from Eric Putt's plant breeding plots at Morden. Using the regular differentials, race group 1 is the only one that I could identify in the 7 or 8 collections that I got from the USSR, from the south Russian areas at Krasnodar and the Kuban Experiment Station. Group 1 is the major one that showed up in Uruguay, although I got race 2 in a few fields. Group 1 is the only one that I have received from South Africa, Southern Rhodesia, Florida, and a few collections from various places in the U.S. until quite recently.

Race group 1, which cannot attack any of the sources of resistance in our differentials, is definitely a combination of entities. In Luciano's territory, at Manfredi in Argentina, they have developed sunflowers with apparently recessive factors for resistance derived from Helianthus argophyllus. I saw the material in the field at Manfredi, and it was definitely resistant when adjacent sunflower varieties were heavily rusted. That same resistant material was completely susceptible to rust, which I identified as race 1, from Pergamino (farther south in Argentina) and from Uruguay. Collections of rust from the field at Manfredi appeared to be race 1 on my differentials. When I inoculated Manfredi sunflower selections with rust from Manfredi, there was considerable segregation, with some of the plants in most lines being resistant and others being susceptible. In other words, race 1 from Manfredi is not the same as race 1 collected in the Province of Buenos Aires in Argentina, in Uruguay, or in Canada. Race group 1 is a combination of biotypes which we cannot distinguish with certainty until someone produces reliable differentials in quantity, and continues to produce differentials with the same phenotype from year to year.

An interesting pattern of host reaction, quite distinct from the "classic" pattern, has showed up several times. I found some wild annual sunflowers growing among the rubble in parking lots in the Loop in Chicago at Christmas, 1955. I collected a pocketful of seed, and tried it out in the greenhouse when I got back to Winnipeg. It proved to be uniformly susceptible to race 1, just like all of the wild sunflowers that I had collected in Manitoba. I was prepared to discard it, but luckily reinoculated some of the plants with other races. Most of the plants were just as susceptible to the other races as they were to race 1. There was some segregation, however. Some plants appeared to be resistant to race 2 or race 3, and in a few cases to race 4. When these same plants were reinoculated with race 1, they were still susceptible to it. This wild sunflower seemed to derive resistance from a completely different gene pool than we had encountered before. This pattern has showed up again in hybrid lines from the Soviet Union, which derived their resistance from the Texas wild annual sunflower.

I made rust readings last week on some lines that Eric Putt had developed from the original Chicago material which I tested in the greenhouse at Winnipeg. Some of the lines show the same striking pattern. Some of the lines derived from the Chicago material include plants resistant to all four races of rust. Similarly, I have encountered plants resistant to all four races in some of the lines developed in the USSR. This material is extremely interesting for use as parental material by the plant breeders to get resistance to all the rust race groups we have already identified. I wish the plant breeders would produce and supply me with reasonably large quantities of differentials with this kind of resistance for use in my work. I am quite sure that sooner or later we will encounter rust collections which can attack this type of resistance. We will be in a much better position to recognize such dangerous rust collections if we have differentials with which to identify them.

I have mentioned that rust from Manfredi behaved like race 1 with a difference. It picked out segregation for resistance in a sunflower population that was uniformly susceptible to the Canadian race 1. Rust was introduced into Argentina relatively late after 1950. Some changes in pathogenicity may have occurred in the few years since then, or the rust may have been introduced from an area with different pathogenicity genotypes than we have so far identified in Canada.

Although the rust collections of the USSR can be identified as race 1 on the Canadian differentials, they also do not seem to be exactly the same as the Canadian race 1. I do not know how long rust has been present on sunflowers in Russia, but it is possible that new pathogenic entities are evolving there.

Rust collections from Texas are certainly going to be different from those we collected in Manitoba. Just last week I finished taking notes on my differentials inoculated with 29 different collections of rust obtained last summer in Texas and Oklahoma by Mr. Luciano. Most of the 29 collections could be assigned quite definitely to race groups 1, 2, 3, or 4, with about the same number in each group. About five of the 29 collections were peculiar, however. Some were like race group 1 with an admixture of other races, and some were like a weak race 4. I have not tried single pustule isolations, because of shortage of time and material. These collections are either mixtures of races, or much more likely, they are distinct entities which we cannot distinguish with sufficient clarity with the differentials I have available. Texas and Oklahoma are recognized as being an evolutionary center for sunflowers. One would expect them to be also an evolutionary center for sunflower rust.

Now, I would like to make some predictions on what is likely to turn up in the future in sunflower rust work. First of all, we know that we have sources of resistance which have not yet been incorporated into commercially available sunflower varieties. I predict without any question whatsoever in my own mind that Eric Putt, and presumably others, will produce varieties with resistance to the races so far identified. I predict equally confidently that we shall eventually find races which will attack those varieties after they are released. The sunflower rust, as you know, goes through its whole life cycle on sunflowers, and goes through it every year. It overwinters in the telial stage, germinates in the spring, and goes through a sexual cycle. The possibility and probability of getting new recombinations is very great indeed. There is a very significant reservoir of rust, and of rust genotypes, on wild hosts. I see the continuation of rust as a major threat to sunflower production for a long, long time to come.

Last week I had a letter from a "tecnico" who had been with me when I looked for rust in Chile in 1954. There wasn't supposed to be rust there. We found it scattered over an area of 500 kilometers from north to south; not, but it was there. It was not found again until Eric Putt looked for it again in 1959-60 and found it. Now they are very glad to

have rust resistant material from Argentina to use in their plant breeding program because they know they have rust in Chile, and they are not likely to get rid of it. Rust was late coming into Argentina; it was later still coming into Chile. I doubt very much if any area with climatic conditions suitable for rust development is going to be safe from the ravages of rust forever or even for very long. The rust is going to get in, but the climatic conditions and the genotype of the host used in the area will determine just how destructive it is.

We have a couple of colleagues from Mexico here. I was very glad to get some Mexican samples some years ago; and if I remember correctly, they included what seemed to be mixtures of races 2 and 3, as well as race 1. Mexico also is an evolutionary center for sunflowers and rust and we can expect all sorts of new things from there.

Eric Putt and his colleague, Dr. Hoes, apparently have evidence for at least five factors or five genotypes for resistance in the host. They can produce differentials with these factors at Morden, and I am quite confident that they will be able to find many more races as long as they have the means of distinguishing them.

I would like to make another comment, perhaps this can come up for discussion later tomorrow, if we have a discussion period. I would like to suggest that it is of value, scientifically and also from the practical point of view, to know what is the potential of the rust pathogen everywhere in the world. The only way we can discover that is to try to get as comprehensive a collection as is possible of rust entities from all over the world, at one place. I would add a further comment that it would be extremely dangerous to try to make such a collection in any area where sunflowers are grown. Because no sunflowers are grown within a hundred miles from where we are at Macdonald, and because we do our work in controlled environment facilities, there would be no danger in maintaining such a collection at Macdonald. Because I am personally very much interested in sunflowers and sunflower rust, I would be only too happy to act as a clearing house for this kind of international determination of sunflower rust if people are interested in sending collections from their areas and if the plant breeders will collaborate by providing the appropriate differentials from time to time. I think that it is something worth doing, and certainly something that I would be interested in doing.

I would like to refer to some other diseases after talking so long about the sunflower rust race picture. I will just mention each of them in passing briefly. I think the next most important disease on sunflower is *Verticillium* wilt, or what I call leaf mottle. Its present distribution is not completely coexistent with sunflowers but is very close to it. I was very happy to be able with coworkers to tie down the cause of the leaf mottle in Manitoba some years ago. I was very embarrassed to meet a woman of 75 in the Soviet Union three years ago who had described *Verticillium* wilt and published the description in detail in an obscure Russian journal

some 20 years earlier. I knew that sunflowers were attacked by Verticillium at the time I was working on leaf mottle, but I didn't know the disease (I had only seen a reference to the article and couldn't get hold of the original) and didn't know it had been described in detail. It is present in the Soviet Union. I know it is present in South America because I saw it there. We have heard about it in various places in the U.S.A. I know it is also present in Africa, and most likely in other parts of the world wherever sunflowers are grown. It is a seedborne pathogen. It is almost certain to be introduced sooner or later wherever sunflower seed is taken and if conditions are favorable it will develop.

One of the interesting things about the Verticillium on sunflowers is that it seems to be quite host specific. I have a couple of students working on Verticillium, not on sunflowers specifically, but on the biology of the Verticillium pathogen and the diseases it causes. One of them has worked a little with the sunflower isolates we have, on other hosts. The sunflower isolate does not attack the other usual hosts of Verticillium to any extent. I cannot say yet that it is a distinct race, but there is specificity there. The Verticillium on sunflowers may be comparable to the one on mint and a few other hosts in that respect. And that I think augurs very well indeed for the production of resistant varieties. Eric Putt has told you that he has Verticillium resistant material; I think that the prospects of continuing to get new resistant material will be very good if and when the pathogen changes. I think that the prospects are also good for reducing Verticillium damage on sunflowers by cultural practices, rotation, etc., as well as by plant breeding.

Another disease which is considered a major one in some parts of the world is Sclerotinia wilt. For instance, they still think it is the most serious disease on sunflowers in Chile. It occurs wherever sunflowers occur. Sclertonia sclerotium is a nonspecific pathogenic organism; it is the same one that rots carrots and many other vegetables in storage and in transit. It causes severe injury to sunflowers in the field as a stalk rot, and it is very important as a head rot. It is the head rot which does a tremendous amount of damage to sunflowers in some seasons in Chile.

Sclerotinia wilt will likely continue to be a problem wherever sunflowers are grown. In areas where it is cool and moist during part of the growing season, especially after the heads are formed, or where irrigation has to be used, the sclerotia in the soil are stimulated to produce apothecia and discharge ascospores. The ascospores blowing around will infect the heads and under such conditions Sclerotinia head rot may be a significant factor.

The possibility of control by resistant lines exists, but I don't think at the moment that we are likely to get a clearcut solution. I think cultural practices are going to be more important for a while. A correspondent has written from Israel about this particular problem, and he says that they are quite prepared to use chemicals. Sunflowers are

sufficiently important to them on his experimental farm (or collective farm) that they are prepared to spend money on possible chemical control for this head rot.

The disease which I think is next in significance is the downy mildew caused by Plasmopara. I believe that the disease is already present almost everywhere that sunflowers are grown. Where downy mildew is not known to exist on sunflowers, I think it just has not been looked for hard enough. There are many areas where I believe it will not be a significant disease because the weather conditions are not favorable for it. But wherever there are relatively humid conditions, while the plants are still in the seedling stage and infection can take place relatively easily, we are likely to encounter downy mildew as a significant factor in sunflower production. You have heard already that there are sources of resistance to downy mildew. As the pathogen is highly specialized on its host, it should certainly be possible to breed for resistance to it and to continue to breed for resistance if the pathogen changes. Cultural practices are also quite important in controlling this disease.

A sunflower disease that we have heard very little about is caused by Sclerotium bataticola. I think there is nothing to worry about from this particular disease on sunflowers in Canada. The pathogen is almost ubiquitious in the soil in warm regions or semitropical regions, places where the winter is mild and where the temperatures go very high during the height of the growing season. It is a very significant factor in parts of South America. It is a significant factor in the Soviet Union. I am quite convinced that it has been mistaken for other diseases in Africa and parts of Asia. As S. bataticola is in the soil just about everywhere in warm regions in the world, I would be very surprised if you did not have it on sunflowers in Texas. (Dr. Kinman: Yes, charcoal rot is common in Texas. It attacks only on the very earliest maturing sunflower.) The disease is particularly bad on maturing and senescent material. I have seen it causing damage on seedlings but in the field it is usually important only on material that is maturing. Sometimes it kills plants prematurely. I think that is a function of specific environmental conditions; heat and moisture stress tip the balance, whether this pathogen will merely be present or whether it will destroy its host.

There is another group of diseases I want to discuss fairly quickly. I will refer to them as minor diseases in this sense. There are many different root rots which can cause damage on sunflower. You have heard references to Fusarium; I am sure there are several species of Fusarium that are significant in various places. I have seen Sclerotium rolfsii causing havoc on sunflowers in South America, and I am sure it would any place else where S. rolfsii is significant. There are quite a few relatively minor stalk and leaf attacking organisms, minor in the sense that they are not likely to be present every year or they are present only in specific localities. You are likely to have them very severe in one country or in one part of one country and not important in others. Certainly we are going to have trouble with them from time to time. Head

rot caused by Rhizopus is not likely to be important in most areas and yet it can be very destructive in some parts of the world in some years.

Lastly, I would like to just lump all the virus diseases. The reason I do this is because we don't know enough yet about the viruses on sunflowers. Almost anybody who wants to study the host range of a virus will inoculate sunflowers in passing, and almost everybody gets positive results. That does not mean that sunflowers are attacked by just about all the plant viruses in nature, but it does mean that they may be attacked at one time or another by various viruses if conditions are favorable. Eric Putt and some of the rest of you have seen what sunflowers look like when they are badly hit by aster yellows. It can be destructive. (I did not bring along any slides of these diseases because usually I show too many slides and this time I was going to be different.) I am quite sure that sunflowers suffer from a number of different viruses. When we know more about all the other diseases which can affect this "most noble crop," we will perhaps have time and energy to work out more information about its virus diseases.

Now, I will conclude with some general statements. First, I can state almost as part of a credo that sunflowers have tremendous potential as a crop. They are being exploited very intensively in some parts of the world for edible oil. Personally, I don't like vegetable oil all over everything, but that is the way people seem to use it in some parts of the world. Where they want vegetable oil, they like sunflowers. It has certainly been very useful also from the farmer's point of view in western Canada in extending the rotation and adding a cash crop.

Disease problems have been limiting factors in sunflower production in the past, and I am quite convinced that they will again be limiting factors in sunflower production in the future. A very few examples: They used to grow sunflowers as a silage crop in eastern Quebec, east of Quebec City, because it is too cold to grow corn there. They had to stop because of Plasmopara. They used to grow sunflowers as a forage and silage crop in Peru. They had to stop because of rust. They are going back into it now that they have rust resistant varieties. The fact that diseases are prevalent or insects are prevalent may put the crop out, or has put the crop out in various places. But, I do believe that most of these major diseases are amenable to control either by plant breeding or by good husbandry. They cannot necessarily be completely avoided or controlled but their effect can be minimized and the crop can be kept profitable and useful by good practice and by the best work of the plant breeders.

And finally, I will state that sunflowers as a crop, and sunflower diseases, require a great deal more research. In the past when I talked about any of my current work on sunflower diseases, it was usually to people who were not deeply concerned about sunflowers, with the exception of the growers. I never thought that I would be called on in North America to talk to a room full of people interested in sunflower research. It has been a very pleasant and stimulating experience. I am sure that the vitally needed research will be greatly expanded as a result of this meeting.

Discussion--

Question: How much does rust affect yield?

Answer: I have results of several years' work, although only abstracts have been published so far. Yield losses with a very susceptible variety, such as S-37-388 in inoculated plots, can be in the order of 75%. Seed increase plots in farm fields in 1951 were completely destroyed by rust. Ordinary susceptible varieties, not extremely susceptible like S-37-388, would quite likely suffer losses of 25 to 50% in a bad rust year. It is important in this connection that resistant varieties, for instance those carrying the 22 factor, show extremely susceptible reaction types when infected by race group 3. or 4. Presumably, if such races became abundant, the resistant varieties would suffer at least as much as the present susceptible ones.

Question: Temperature and humidity are important in rust development on small grains. Does higher humidity lead to heavier rust infection on sunflowers?

Answer: Yes, the weather may only have to be favorable in a small area for sunflower rust to develop, however, as compared to cereal rust. As you know, cereal rust outbreaks in western Canada depend on weather conditions being favorable all the way from north Mexico and Texas early in the spring, and in the successively more northerly states as the season progresses. If it is too cold or too dry anywhere along the line, western Canada may escape rust even if the varieties grown are susceptible. This is not true for sunflower rust because the spores do not have to "migrate" to overwinter. The telial stage overwinters on sunflowers, germinates in the spring, and can reinfect susceptible sunflowers if any are nearby. Therefore, the conditions in Texas or Oklahoma or Kansas are not important.

Isolation is much more likely to be important in protecting sunflowers from rust than it would be for cereals. Even in 1951, when rust was heavy and extremely widespread on sunflowers in Manitoba, there were isolated fields right in the central sunflower area which had little or no rust on them, thanks to a distance of a mile or so from the nearest field which had rust the previous year, or to a good shelter belt of trees around the field, or similar isolating factors. Even though the sunflower crop was extremely concentrated in the Red River Valley, the total area grown was relatively small, nothing like the tremendous expanse with wheat and wheat rust.

The situation is quite different in South America. In 1957 I found no rust on sunflowers in Uruguay when I looked for it sometime early in January. A few days later I had a letter from people in Pergamino, Argentina, saying that rust was becoming quite general on sunflowers in the area. Shortly after we had a "pampero" the typical wind from the south and west from Argentina. About two weeks after that I could not

find a field in my survey that didn't have scattered pustules of rust on almost every plant. There was such a tremendous population of rust spores in the air that it blanketed the whole country. This situation does not exist in North America because sunflowers are not grown on such a scale. The spores certainly seem to be at least as well equipped as those of the cereal rusts to withstand long distance travel in air masses; they are more resistant than cereal rust spores to low temperatures which might be encountered at high elevations.

Question: Are there any important diseases caused by bacteria that you have worked with or know about? Are nematodes a problem on sunflowers?

Answer: I have not worked seriously with any bacterial diseases on sunflowers, although I have seen bacterial stalk rots. I do not know about any significant sunflower diseases caused by nematodes, although they may occur.

Question: We have had at least 390 sunflower introductions from 23 countries since we started our work, and we probably still have seed of about 100 of these. This seems like a real opportunity to use introduced germ plasm in breeding for resistance to diseases and other problems.

Answer: Wherever such sunflower introductions are of interest, they should be screened for reaction to local rust races and other pathogens. I would be quite prepared to screen them in detail if anyone is interested. Incidentally, now that we know how extremely heterogeneous and heterozygous our sunflower lines are, we have developed more critical tests for rust reactions. I used to inoculate ten or twenty plants of a selection with each of the races I was using and assumed that I would get reliable results. We no longer depend on this "statistical" approach. We put eight different isolates on one pair of leaves. Every plant is inoculated with our four "Canadian" rust races, to make sure that we can identify the rust-reaction phenotype of the individual plant. In addition, each of the plants is inoculated with three or four or sometimes five unknown races so that we can compare them with the reaction of the known races on the same plants. This is obviously a very tedious and time-consuming program, but I would be prepared to do this kind of inoculation to a limited extent with important material.

You would have no hesitation about bringing in sunflower germ plasm from any part of the world to Texas or to Manitoba provided it had gone through a sanitary screening process first. Dr. Leppik handles all the introductions in the U.S., and he is quite prepared to certify that material that he has increased is free of disease. It is then safe to grow it wherever you wish. But it would certainly not be wise to introduce new and potentially dangerous rust collections from other parts of the world to this area in Texas, or to Manitoba, or to any area where sunflowers are grown or are important.

I believe that the cereal rust workers study the potential of dangerous foreign cereal rust races on the Virgin Islands, which are so isolated that the rust cannot get to the North American mainland. We

have the practical equivalent of that at MacDonald College. We are on the island of Montreal with no sunflowers being grown commercially anywhere in the area. We do our determinations in the greenhouse in the winter and in growth cabinets in an underground room in the summertime. We could test rust from any place in the world without posing any threat to sunflower production in North America. This is the sort of critical testing that I indicated I was interested in doing, although I would, of course, be interested in trying out a good representative world collection to see what kind of resistance I could encounter there.

Question: When my sunflower plants were only about an inch high, I found three with rust on them.

Answer: What stage of the rust was it, uredospores or aeciospores? Were they the typical brick red pustules? Was the seed sown in the spring or were they volunteer plants?

Question: This was material which had shattered from susceptible varieties in the previous fall, but the plants emerged in the spring. They were uredial pustules, present on plants about an inch high.

Answer: I have heard reports of uredospores of sunflower rust being transmitted with the seed. I haven't seen it myself, but that does not prove it doesn't happen. It is possible that in the case you describe there may have been a good accumulation of rust spores with the volunteer materials. Some of these uredospores may have survived over winter and attacked the seedlings just as they came out of the ground. I think that there is a possibility of this happening, although I do not know how high the probability would be.

Question: Where does the aecial stage occur on sunflowers? I have never seen it.

Answer: Usually the first pycnia and aecia are on the cotyledons of volunteer seedlings. In 1951, which was a rust disaster year on sunflowers in Manitoba, we found pycnia and aecia on the cotyledons of volunteer sunflower seedlings early in June. I can't be sure - 13 years later - about the dates, but I think that there was secondary uredial infection present by about the middle of June. The pycnia and aecia which followed them were started by spores produced by germinating telial spores on sunflower stalks, and to a lesser extent on leaves, which had not been completely covered by soil and had lived through the winter. They are much more likely to survive on the stalks which are more resistant to mechanical breakdown and decay, but they can also survive on the leaves.

Teliospores in Manitoba do not germinate very well unless they have been exposed to freezing temperatures. This is not true everywhere. I got down to Uruguay in July in the middle of winter in 1956. They had sown sunflowers very late so that living plants would still be present

when I arrived. There were adult plants in the plot with the upper leaves completely covered with rust. Scattered among the telial pustules were pycnia and some aecia. Those teliospores had germinated in situ. They had not been exposed to frost, but they were apparently adapted to existence without frost and had germinated. We can do this in the greenhouse with some success, but it does not happen in the field in Manitoba. It apparently happens in South America.

Question: Are there any wild annual sunflowers in Quebec?

Answer: They have been reported, but they are scarce.

Question: Is Plasmopara likely to spread?

Answer: I am not worried about the spread of Plasmopara from my test material. In all the years I made surveys of sunflower disease, only once or twice did I see secondary infection on leaves from sporangiospores of Plasmopara. All the infection we found in Manitoba was systemic from inoculum in the soil. Even if conditions favored secondary spread by sporangia from leaf to leaf or plant to plant, it would not pose any danger at MacDonald College. We work in the greenhouse during the winter. In spring long before there is any amount of growth outside it gets too hot to work in the greenhouse so we move our studies down to the controlled environment cabinets underground.

Question: Do you work with Plasmopara in the field?

Answer: Yes, but only with infested soil collected in the Province of Quebec.

Question: Have you worked on the host range of the sunflower isolate?

Answer: Not yet, although we are going to do that. I would like to get as many collections of Plasmopara from distinct geographical areas as possible to compare them in one place. From my own experience I am not inclined to agree with the Russian workers (who are "splitters"), who believe that their isolate is a separate race or perhaps even a separate subspecies.

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