DISCUSSION

Chairman :

On behalf of this Symposium I wish to thank Professor Martin. I also take this opportunity to make two comments and to ask Professor Martin two questions.

My first comment is that, from my own preliminary observations on sweet potato diseases, it would appear that our main problems in the Caribbean are caused by **C. fimbriata** and **Rhizopus** species. Of these the black rot disease caused by **C. fimbriata** is by far the more important.

My other comment is linked with two questions. The present emphasis on the economic need to increase food crop production in the developing countries may well lead to an increase in sweet potato cultivation. Professor Martin, you have indicated that at present the level of resistance to black rot in sweet potato is unlikely to be adequate for economic control of the disease. The pathogen, **C. fimbriata**, is already widespread in the Caribbean islands, and black rot accounts for very heavy losses. The level of soil contamination seems likely to increase, first, because there is no climatic extreme such as winter to help kill the pathogen ; secondly, because some territories practise unbroken sweet potato cultivation on the same land ; and thirdly, because of inter-island trade. Further, our harvesting and post-harvest handling methods for sweet potatoes are still far from satisfactory. This increases proneness to tuber infection.

Professor Martin, would you like to comment on the likelihood of black rot to increase in importance in this area ? And would you also kindly give us a little more detail on the fungicidal treatment and hot-air treatment ?

W. Martin (U.S.A.) :

In the United States frost helps to kill off both pathogens and insect pests of sweet potatoes. I appreciate the problems you have here, because you do not have winter, but I am still optimistic about the possibilities of controlling black rot here.

With regard to resistant varieties, we are still searching for higher levels of resistance, but I do not wish to minimise the importance of the resistance to black rot that is at present available in some sweet potato selections. I would certainly recommend the planting of these varieties.

Concerning fungicidal control of black rot I wish to say that pre-planting chemical applications constitute a promising control measure. This method costs about $8.00-$10.00 U.S. per acre and is therefore economical. Unfortunately, we have nothing as effective against black rot as 2,6-dichloro-4-nitroaniline is against soft rot. Although there are many materials that will kill the black rot fungus and prevent secondary spread onto the root, most of these materials produce a burning type of injury and cause the sweet potato tuber to look even more unattractive than when attacked by the black rot fungus. There is not a big enough margin between the fungicidal dosages and phytotoxic dosages. I think, however, that it is desirable to screen many of the newer chemicals.

E. Trujillo (Hawaii) :

Professor Martin, is there any evidence that insect vectors are involved in the transmission of **C. fimbriata** in the black rot disease ?

W. Martin (U.S.A.) :

I think Dr. Iton should answer that question as he has told me of instances of insect transmission of **C. fimbriata** in other crops.

E. Iton (Trinidad) :

C. fimbriata is transmitted by insects in several other plant diseases. From my

limited observation in the Southern Caribbean, there appears to be no insect transmission of **Ceratocystis** as a pathogen of sweet potatoes in the field. I also have no indication of insect transmission of the pathogen among stored sweet potatoes, but I am fairly confident that there is transmission of **Ceratocystis** by mites within the sweet potato stores. There is a high population of mites in most sweet potato depots and **C. fimbriata** sexual spores, which are discharged in a mucilaginous matrix, do adhere readily to the bodies of these creatures.

B. Williams (Trinidad):

Dr. Martin, in these parts we have had many reports from different sources that clonal deterioration occurs in our sweet potato varieties. Indeed, our breeding programme has been criticised in some quarters on this account. It has been said that we release our new varieties too early. This criticism is, however, mere speculation. Nevertheless, there is a well-documented case of this decline in yield. Professor Harland, perhaps one of the earliest sweet potato breeders in the world, early in this century developed a variety, V 52, in St. Vincent. This is a good variety and the annual reports of the Departments of Agriculture show that it has been included in most trials throughout the years, but a decline in yield can be detected in it.

Recently we sent material from all sweet potato varieties collected at St. Augustine for investigation by Sir Fredrick Bawden at Rothamsted, England. He informed us that checks under the electron microscope had shown no evidence of virus particles in the material. He has plans, however, to test extracts against test plants to confirm whether viruses were absent.

Professor Martin, do you think that the clonal deterioration reported in this region could be explained by reference to (1) your observation of nematode population build-up in cultivation over the years and (2) Dr. Iton's point in the opportunity for pathogen build-up to occur in these areas?

W. Martin (U.S.A.):

First, about the viruses. The aphid-transmitted viruses are very unstable. At present a virus expert and I are trying to find some way to stabilize these viruses so that we can see them under the electron microscope. So far the only ways by which we can detect them are by symptoms manifested in the plant and by transmission tests. I am therefore not suprised that examination with the electron microscope revealed nothing.

With regard to nematodes we would have to investigate the soil to determine which species are present. For example, the reniform nematode, is an extremely destructive parasite of sweet potato and I understand from one of your staff members in this institution that it is present in some of the soils here. This nematode may well be causing your varieties to deteriorate.

R. Barnes (Trinidad):

Professor Martin, you mentioned that the factors determining resistance against the reniform nematode and the root knot nematode appeared to be different. Is this conclusion based on observations on varietal selections? Do varieties either go down to one or the other nematode, or has work been actually done on the factors which govern resistance?

W. Martin (U.S.A.):

I should have put quotation marks around the term factors. This conclusion is based on the fact that our root-knot-resistant varieties are very susceptible in most cases to the reniform nematode and the varieties most resistant to the reniform nematode are actually most susceptible to the root knot nematode.

E. Gooding (Barbados):

Professor Martin, we saw in the field the other day Irish potatoes affected by

Sclerotium. Do you think that 2, 6-dichloro-4-nitroaniline would be successful against this fungus on Irish potatoes ?

W. Martin (U.S.A.) :

Was it attacked by **Sclerotium rolfsii?**

E. Gooding (Barbados) :

I am not sure, Mr. Haynes described it as **Sclerotium,** he did not specifically say **Sclerotium rolfsii.**

R. Pierre (Trinidad) :

I should like to confirm that the species involved in the Irish potato disease is **Sclerotium rolfsii.**

W. Martin (U.S.A.) :

This may be an extremely interesting chemical to try. The sweet potato itself can take concentrations of 600 lb. of active material per acre of this chemical and not show any evidence of phytotoxicity.

E. Gooding (Barbados) :

Would it be desirable to treat the potato before planting ?

W. Martin (U.S.A.) :

This is our approach to disease control in our sweet potato seed beds. The chemical has a life of about ten to fifteen days but protection may be necessary over a longer period of time. Of course, it is possible to increase the rates of application but the cost of this chemical at rates high enough to control **Sclerotium** in a crop like Irish potatoes over a long period of time will be prohibitive.

E. Gooding (Barbados) :

Professor Martin, I wonder whether the internal spotting of yams which I described yesterday is similar to the internal cork of sweet potatoes which you depicted in your photographs ?

W. Martin (U.S.A.) :

This condition in yams is very interesting and I would certainly like to see it investigated. It would also be interesting to determine whether the white fly-transmitted viruses in many other tropical plants are the same types that are found in sweet potatoes.

S. Harland (Peru) :

May I be permitted Mr. Chairman to make a short comment on a comment. Regarding the question of degeneration in sweet potatoes, I think the important point to emphasize is that the sweet potato, being a high polyploid, is an intensely mutable product and we know, from many other sources, that mutation in minute physiological and morphological characters takes place in many organisms with a velocity many times greater than that of the gross morphological characters that we observe. There are two kinds of 'mutations' — the hidden or invisible type which leads to very rapid changes in the adaptive complex (of course, yield is one of those things) and the degeneration through virus. Now, I think myself that the sweet potato is a plant which is admirably suited for intensive study from this point of view. I think the point about degeneration is well taken, I feel quite convinced that clones may rapidly decline from a very high yield when they are new to a position of stability as the years go by.